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CANCER OF THE THYROID GLAND*

HOWARD M. CLUTE, M.D.
AND
LAWRENCE W. SMITH, M.D.
BOSTON

From 1916 to 1927, 3,389 patients were operated on in the Lahey Clinic for diseases of the thyroid gland. Of this number, sixty-seven had malignant disease of the thyroid, an incidence of 1.68 per cent. In fifty-four of these sixty-seven cases, the condition was proved to be malignant by the microscopic study of the sections removed, and the remaining thirteen cases were diagnosed as hopeless and inoperable malignant disease by clinical methods alone. A similar incidence of malignant disease of the thyroid was reported by Graham, who said that slightly less than 2 per cent of the thyroids examined in the Lakeside Hospital Laboratory showed malignant disease. Craven stated that from 1 to 5 per cent of all thyroids operated on show a malignant condition, according to the reports of various observers. Pörtmann in the Cleveland Clinic reported an incidence of 1.6 per cent of malignant disease in persons with disturbance of the thyroid gland, and Eberts and Fitzgerald gave 1.8 per cent as the rate of occurrence in 612 operative cases of thyroid disease.

SEX

Of the sixty-seven patients in this series, fifty-eight were females and nine were males, a ratio of 6.4 women to 1 man.

AGE

The youngest patient in the group was a girl of 20 and the oldest, a woman of 82. Figure 1 shows the relationship between the number of patients and their age. It is to be noted that only six of sixty-seven patients were below 41 years of age. Of these six patients, however, two are dead, one is not traced, two have been well for five years or more, and one has been well for six months since operation. While it is obvious that the greatest age incidence of malignant disease of the thyroid is between 50 and 65, it is equally obvious that the youth of the patient is no proof against the chance of its occurrence.

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* From the Lahey Clinic and the Pathological Department of the Medical School of Harvard University.

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GOITER

A goiter had undoubtedly been present in the great majority of all our cases before the malignant condition was found, in one patient for forty years. In other cases, goiter had not been noted until two weeks before admission to the clinic. In most cases the goiter was adenomatous, as described by the clinical examination. Histologically, of the fifty-four specimens examined we could be certain that the malignant condition arose in a preexisting adenoma in forty-three cases, and in eight other cases it seemed probable that the premalignant lesion was an adenoma. In three cases it was impossible to state the previous lesion. It seems fair to state, therefore, that in fifty-one cases (94.4 per cent) an adenomatous goiter preceded the malignant goiter.

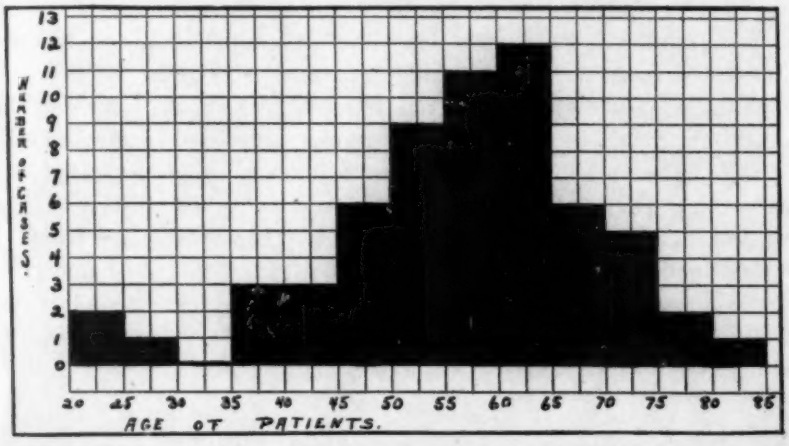

Fig. 1.—Age incidence of malignant disease of the thyroid in this series. Although most cases occur between 50 and 65, it is apparent that cancer of the thyroid may occur at any time in adult life.

Graham reported that at least 90 per cent of his cases of malignant disease of the thyroid arose in a preexisting adenoma and that in many of the remaining 10 per cent he was unable to state that they did or did not arise in an adenoma.

PATHOLOGY

The classification of cases of malignant disease of the thyroid is among the most difficult problems of differential diagnosis for the clinician and pathologist alike, in many instances.

We have attempted to formulate a working classification of the various types encountered, especially in respect to the prognosis. That there are variations within these groups is no doubt true, as might be emphasized by a review of the considerable literature on this subject;

conversely, however, it is equally true that within the individual groups the cases have shown an amazing uniformity in their course.

In reviewing the literature, one is struck by the multiplicity of terms applied to the tumors of the thyroid just as one is struck by the confusing nomenclature of the hypertrophies and hyperplasias of the gland. Anything which will be a step toward the simplification of this problem seems to be a justifiable task. After a perusal of our own material and the literature, it seems highly doubtful if such a thing as primary sarcoma of the thyroid exists. That a sarcomatous degeneration of the stroma of thyroid tumors may occur is, however, not to be denied. This view is one which Ewing has expressed in his book on neoplastic diseases, and the more one studies the available material, the more probable this point of view becomes.

Grouping of Cases

Histologic Groups	Females	Males	Totals	Dead	With Recurrence or Dying	Not Traced	Living and Well	Duration
I. Malignant adenoma								
A. Papillary adenoma..	14	2	16	4	2	2	8	4 over 4 years (6, 5, 4½, 4) 3 over 1 year (2, 2, 1½) 1 under 1 year
B. Fetal adenoma.....	15	2	17	7	3	1	6	2 over 4 years (4½, 4½) 4 over 1 year (2, 1½, 1⅛, 1⅛)
II. Squamous cell carcinoma	..	1	1	1	..	..	..	
III. Giant cell carcinoma.....	8	1	9	8	1	..	..	
IV. Small cell carcinoma.....	10	1	11	8	1	2	..	
Patients not operated on.....	11	2	13	10	1	2	..	
Totals.....	58	9	67	38	8	7	14	6 over 4 years 7 over 1 year 1 under 1 year

We have adopted a grouping of the cases which is hardly worthy of the term classification, but which tends to separate under a few major headings practically all the tumors which one encounters. This grouping is shown in the accompanying table.

Most of the tumors arise from adenomas of one type or another. Our major group is made up of the malignant adenomas: (1) the papillary adenocystoma type and (2) fetal adenoma type. Under this term we have to recognize two forms of the tumor. The first is a tumor of a papillary cystadenoma type which ultimately becomes malignant. These are among the most difficult of the thyroid tumors in which to recognize beginning malignant disease, as the borderline between simple papillary hyperplasia and a true malignant condition is almost indeterminable. We may compare them closely to the papillary cystadenomas of the ovary or the mucous polyps of the gastro-intestinal tract.

Of the fifty-four cases, sixteen, or 29 per cent, fall histologically into the group of malignant adenomas of the papillary adenocarcinomatous

type. Fourteen of these cases were in women and two in men. The average age in this group was 56 years, the oldest patient being 80 and the youngest, 20. The average duration of the goiter before operation, when known, was 10.1 years, but this average would be considerably increased if the exact duration in three of the cases in which the goiter was known to be present for years could be included in the figures. The shortest duration of the goiter before operation was one month and the longest duration thirty years. Four patients of this group were dead at intervals of from three months to six years after operation. Two patients had a recurrence of malignant disease, in one case at six months and in the other after five years. Two cases cannot be traced. Of those remaining, eight patients are living and well at intervals of from one to six years after operation.

Microscopically, the criteria of malignant disease are those of malignant disease elsewhere: anaplasia, mitosis, invasion of the capsule and lymphatic extension. All of the sixteen cases of this group show such invasion of the capsule.

Interestingly enough, it is the type of tumor which responds best to irradiation. This in turn has some tendency to discount our histologic interpretation, as the clinical results are more strikingly successful. Furthermore, these tumors are apt to be large enough to be noticed relatively early in their course; and accordingly operation is performed earlier and on younger patients; so that the chance of their complete removal and of their removal before marked malignant degeneration occurs is much better. We feel that the evidence is sufficiently complete, however, to suggest that if let alone all of these tumors will undergo malignant change, sooner or later, so that their removal at the earliest possible time is absolutely indicated.

There is considerable evidence to support the view that these tumors may arise from the so-called fifth pouch or posterior outpocketing of the pharynx. It has been shown in another paper that the lateral aberrant tumors of the thyroid are derived from this group of cells in their migration and fusion with the main body of the thyroid gland.

In the second form of the malignant adenomas, one is dealing with an entirely different type of case which arises from the so-called fetal adenomas. The variation in form is almost endless, the tumors varying from those made up of anaplastic-appearing trabeculae and sheets of undifferentiated epithelium showing little if any acinar formation to tumors composed of large colloid distended acini, for the most part. Somewhere midway between these two extremes lie the majority of the tumors; they are relatively cellular and are made up of cuboidal epithelium forming minute acini but containing little if any colloid; they are usually complicated by cystic degeneration of the stroma.

Of the fifty-four cases in this study, seventeen, or 31 per cent, fall into the malignant adenoma group arising in fetal adenomas. Fifteen of these patients were women and two were men. The average age of the patients was 50.6, the youngest being 25 and the oldest, 69. The average duration of the goiter before its removal was eight years, varying from two to twenty years in different cases. Of these seventeen patients, ten are now dead or dying, with recurrence of the disease at

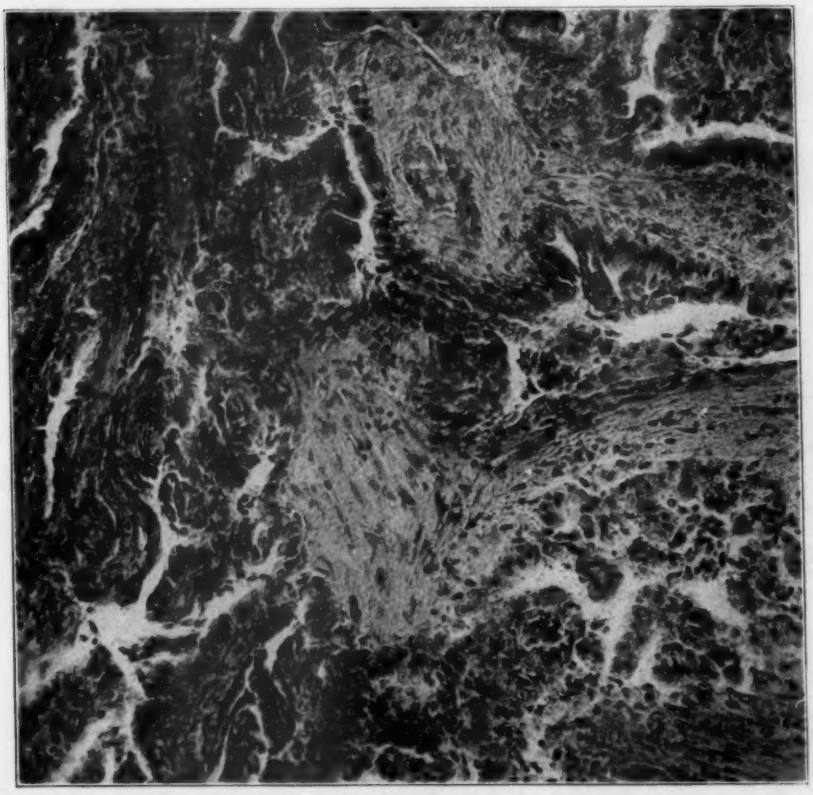

Fig. 2.—Papillary adenocarcinoma illustrating characteristic invasiveness of tumor, with disorderly appearance of epithelium. Low power. Leitz, 16 mm. obj.; ocular, 2x; magnification approximately × 100.

intervals of from six months to four years after the operation. One case is not traced, and the remaining six patients are living and well at intervals of from one to four years. Clinically, we are unable to distinguish this group by any criteria to be noted in either history or physical examination. Malignant degeneration in these patients can rarely be suspected from the gross appearance of the adenoma at operation unless the process has obviously invaded the capsule of the adenoma. We have learned that in this group of tumors the usual criteria of

malignant disease—mitosis and anaplasia—are of little diagnostic or prognostic value. Graham's invaluable contribution emphasizing the importance of invasion of the blood vessels in these tumors cannot be too thoroughly applauded. Indeed, it is more often than not the only criterion of value. We frequently see adenomas which on histologic examination appear to be potentially extremely malignant but without clinical evidence of a malignant condition, and, on the other hand,

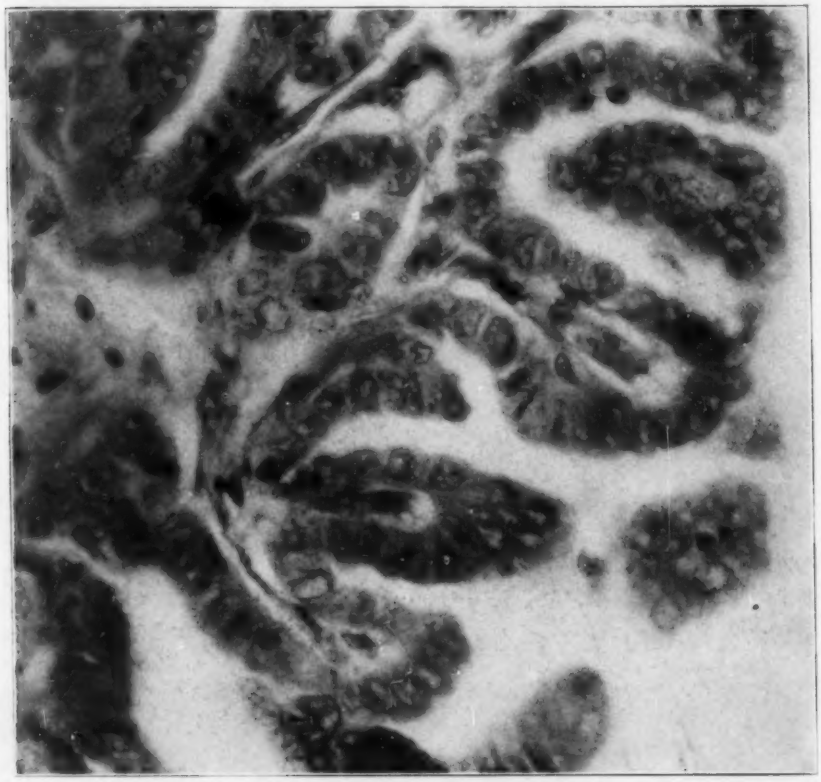

Fig. 3.—Papillary adenocarcinoma showing typical arrangement of cells on papillary stalks, regular appearance of cells, and low grade malignant disease. The mitotic figures should be noted. High power. Leitz, 4 mm. obj.; ocular, 2x; approximate magnification, × 1000.

adenomas which appear essentially harmless under the microscope but which have already metastasized. In one instance, a malignant adenoma as established by Graham's criterion of vessel invasion was removed from a woman, aged 25. Other histologic indication of malignant disease was not present. The adenoma had existed at least six years before the operation, and four years have elapsed postoperatively without recurrence. Such a case as this, therefore, makes one question whether

even this criterion of invasion of the blood vessels always warrants the diagnosis of malignant disease.

We feel that all these extremely cellular adenomas are potentially malignant and should be removed surgically to prevent malignant degeneration with invasion of the blood vessels and distant metastasis. It is usually impossible to tell clinically before operation what the histologic picture of any given adenoma is going to be. We are convinced that it is never justifiable to incise such a nodule for biopsy, because of the

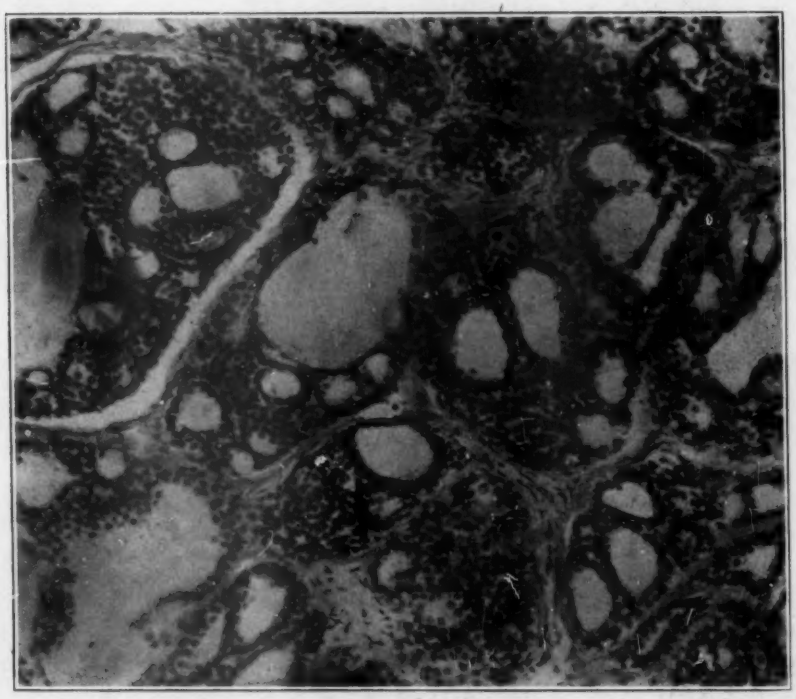

Fig. 4.—Fetal adenoma, illustrating innocent appearance of this type of tumor which, however, was the cause of death from metastasis via the blood stream. Low power; approximate magnification, × 100.

ease with which these tumors seem to gain access to the blood stream. And neither is it safe not to treat persons who have such nodules, no matter how long they have been present. We have seen several instances in which adenomas that had existed for from twenty to forty years without symptoms or change in size ultimately progress to malignant degeneration and either local extension or distant metastasis. Neither the surgeon nor the pathologist has any doubt about many of the tumors; many of them, however, present much difficulty in diagnosis to both the surgeon and the pathologist.

Here, again, it is apparent that the only rational therapy as insurance against subsequent malignant disease is the operative removal of any adenomatous nodule. From a practical point of view, the chief difficulty to be encountered in this regard is the differential diagnosis from the so-called multiple colloid adenomatous or nodular goiter, which is not in any sense a neoplastic condition but merely one of irregular functional hypertrophy, hyperplasia and involution, either spontaneous or

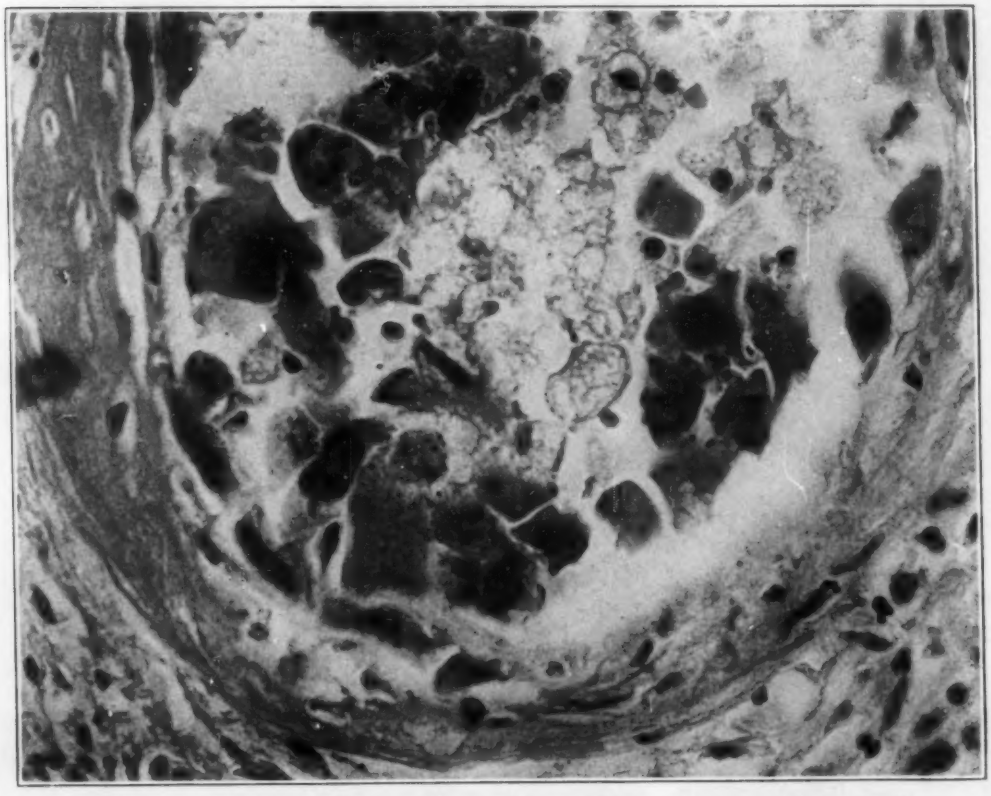

Fig. 5.—Invasion of the blood vessels by adenocarcinoma. High power; approximate magnification, × 1,000.

influenced by iodine therapy. Such nodular goiters are equally if not more susceptible to formation of true adenoma.

The second group of tumors is an extremely rare one, of which we have but one example. That is the true squamous cell carcinoma, arising usually from the thyroglossal duct (fig. 6). In other tumors, we frequently find a tendency for the cells to assume a pseudostratified or even spindle form, but in no other type do the other characteristic histologic criteria hold true. The course is that of epidermoid cancer elsewhere—lymphatic extension and death from metastases—complicated

by mechanical symptoms of pressure. The single case in this group was in a man, aged 56, who died fifteen months after the appearance of the tumor was noted, with extensive glandular metastases to the axilla and the posterior cervical region. Intensive x-ray therapy did not have any influence on the course of the disease; in fact, the tumor seemed to progress with even greater rapidity after the first x-ray treatment. This may well be merely a coincidence.

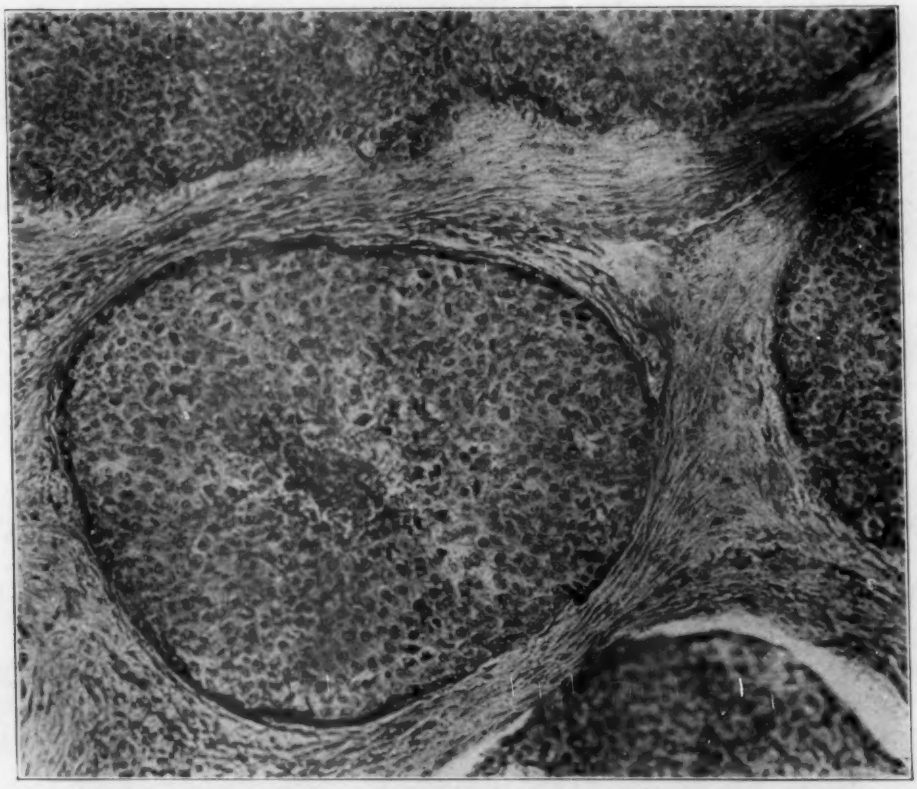

Fig. 6.—Squamous cell carcinoma. The islands of epithelium in the fibrous stroma, which are comparable to similar tumors of keratinizing epithelial origin, should be noted. Approximate magnification, × 200.

The third group of tumors is an even more frankly malignant type with an almost invariably fatal course within from six months to a year. This group can be subdivided somewhat artificially into two major types—first, that in which the carcinomatous picture predominates, and second, that in which a sarcomatous picture, microscopically, is more prominent. These tumors can all be called giant cell carcinomas to distinguish them from the other varieties of thyroid tumors.

There are nine specimens in the fifty-four (16.6 per cent) that were grouped as giant cell carcinomas. There were eight females and one

male in this group. The average age was 60 years, the oldest patient being 72 and the youngest, 46. In three of these nine cases a goiter had been present for several years which within a month before operation had grown rapidly in size. In the remaining six cases, goiter had not been noted until two months or less before operation. Eight of these nine patients are dead, and one has a recurrence. Seven of the eight who died lived less than four months after operation, and the eighth patient lived but one year.

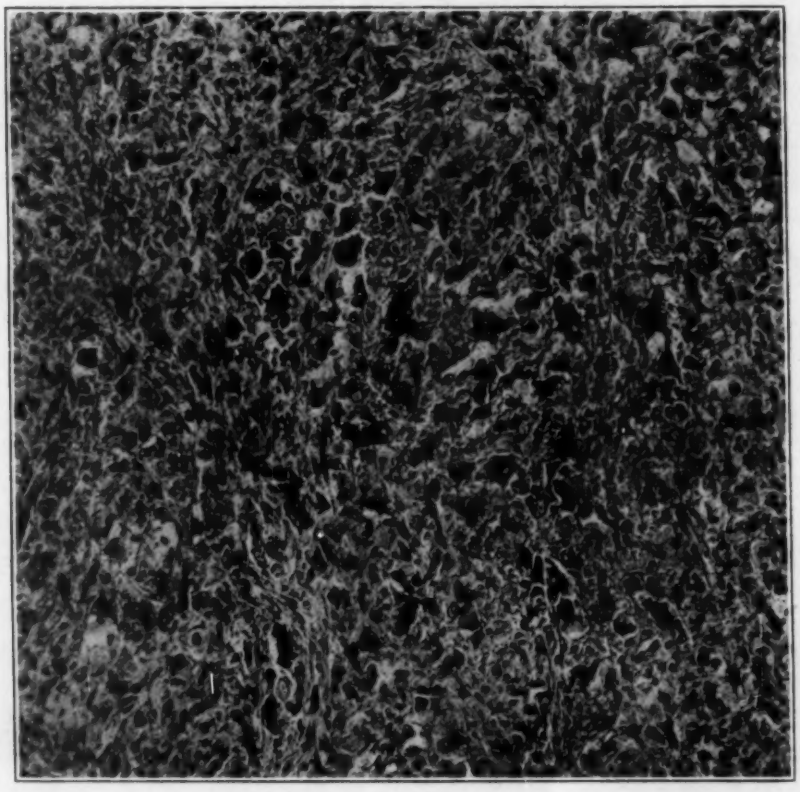

Fig. 7.—Giant cell carcinoma, illustrating striking similarity in appearance to the usual fibrosarcoma. The occasional tendency toward alveolar formation should be noted. Tumor giant cells, multiple mitoses, etc., are present. Approximate magnification, × 100.

The essential histologic structure is that of a rapidly growing tumor with many large multinucleated giant cells. They tend to be relatively vascular tumors with a great deal of hemorrhage and necrosis. A perivascular arrangement of the cells is not at all uncommon as in other pseudosarcomatous tumors. Many of these tumors unquestionably arise from adenomas as do the others, but at the time of operation they have

become so diffuse that any attempt to prove this origin is futile. In some cases the picture is one of diffuse bilateral general involvement of the gland. It is probable, however, that even if not strictly adenomatous in origin they are unicentric, although even this is difficult to prove. They invade the adjacent tissues widely, especially seeming to involve the great vessels of the neck, particularly the jugular veins. They tend to remain relatively localized until invasion of the blood vessels has occurred, when they may set up distant metastases.

The particular point of interest in this group is their close resemblance to fibrosarcomas, and indeed from single sections it may be impossible to recognize their epithelial or even thyroid origin. Certain of the tumors show well (fig. 7) the transition from polyhedral atypical epithelium to spindle cells with definite resemblance to connective tissue. One of the chief difficulties lies in the fact that the stroma of the tumors unquestionably undergoes malignant degeneration; in some cases, as in tumors of other organs, for example, the breast, the sarcomatous degeneration of the stroma is a more prominent feature than the epithelial element of malignant disease. This may lead one to make the diagnosis of sarcoma. This is justifiable, perhaps, on the basis of the histologic criteria, but as viewed from the general hehavior of the tumors and a more thorough study of the cases microscopically, their primary epithelial character is usually brought out and the diagnosis of carcinoma established. Clinically, this type of cancer can be suspected when a long-standing goiter suddenly shows rapid growth. The appearance and rapid growth of a goiter in a person 60 years old likewise suggest this type of tumor. Operation is of no avail, and roentgen treatment has not given any benefit. This group is marked clinically by the sudden onset, rapid course and invariably fatal outcome of the disease in spite of any available treatment. Operative procedure in this type of tumor is justified only in establishing a diagnosis and in securing relief from mechanical pressure symptoms—a palliative measure of relatively short value.

In the fourth and last group of cases, we have another debatable form of tumor. For purposes of classification, we have designated it as the small cell carcinoma. This is the type of tumor which is sometimes called lymphosarcoma, as it is made up of small round cells, poorly differentiated, as a rule, with a prominent nucleus and an almost negligible amount of cytoplasm. The chief differential features histologically are the tendency here and there to form acini and the fact that the chromatin of the nucleus is not arranged typically as in the lymphocyte. In addition, we can in some cases find the transition of the tumor from an adenoma. It does not particularly matter what term we apply to this group. It seems to us that the important point is to recognize it as a distinct type of tumor.

Eleven of the fifty-four patients (20.5 per cent) fall into the group of cases of small cell carcinoma. Ten of these were women and one was a man. The average age in this group was 61.9 per cent, the youngest patient being 48 and the oldest 72 years. The average duration of the goiter before operation was four months, varying from one and one-half to fourteen months. Of these eleven patients, two could not be traced, eight are known to be dead at intervals of from one month to

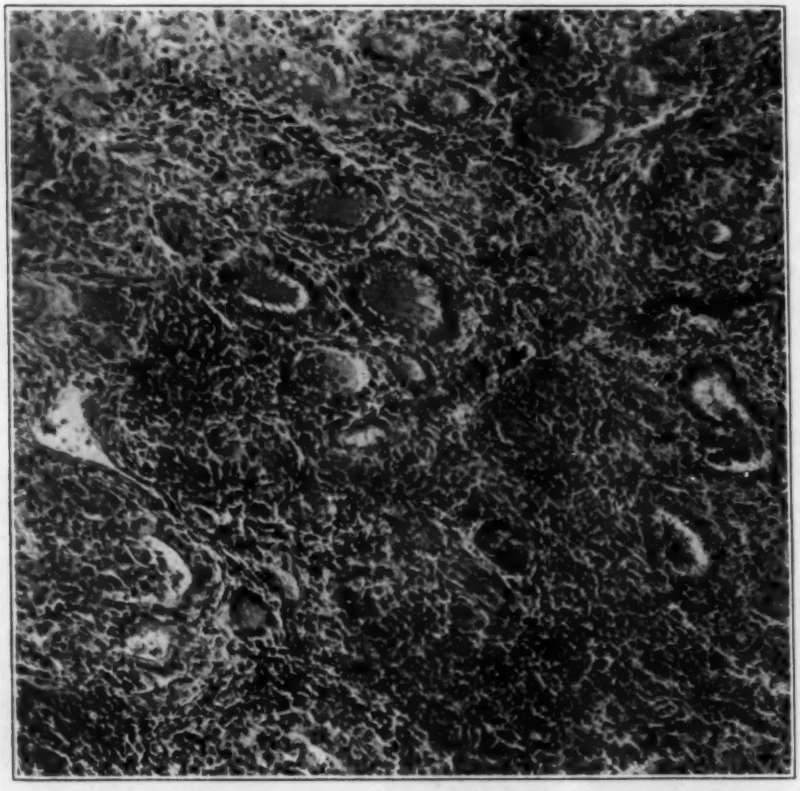

Fig. 8.—Small cell carcinoma, illustrating a typical field showing invasive character of the tumor, not all the acini being yet involved but being destroyed by the advancing tumor. Other areas clearly show transition from normal to tumor cells. Compare the picture of chronic ligneous thyroiditis with round cell infiltration. Low power; approximate magnification, × 100.

two years after operation, and the remaining patient had a recurrence of the disease within eight months of the operation.

Microscopically, these tumors are among the most difficult to identify definitely. They are the type sometimes considered as lymphosarcomas, but we are firmly convinced of the epithelial nature for reasons given elsewhere in this paper. The chief differential point histologically is

from Riedel's struma, with which they may be readily confused. They are characterized by diffuse infiltration, either of a single lobe or of the entire gland, by small round cells which here and there show a definite tendency toward lumen formation. There is usually evidence of marked local invasion of the surrounding tissues, particularly the vessels of the neck and occasionally the smaller vessels of the gland itself. This resembles the condition found in the malignant fetal type of adenoma. Clinically, it is impossible to distinguish this group. The thyroid gland is diffusely involved and is firm and fixed. Many patients in this group may be suspected of having severe thyroiditis (Riedel's struma). The differential diagnosis between small cell carcinoma and Riedel's struma is often impossible, and operation is necessary to determine this point. In two of our cases we originally considered the condition histologically as atypical strumitis rather than tumor, but it was proved subsequently to be small cell carcinoma by recurrence of the tumor. From the patient's point of view the differential diagnosis is of great significance, for in Riedel's strumitis she may well expect a favorable end-result, while in the small cell carcinoma the prognosis is bad. This is an important group, then, in which to do a biopsy to establish the diagnosis, and it is a group, unfortunately, which is not usually seen clinically until relatively late in the course of the disease, since there is but slight enlargement of the gland. Histologically, it shows a marked tendency toward vessel ingrowth, but only terminally does it produce distant metastases—in one case, of the lung, liver, kidneys and bones, as proved by autopsy. The course of this type of malignant disease is rapid and probably always fatal. Roentgen treatments have not been of any benefit.

SYMPTOMS AND COURSE

In this group of sixty-seven cases there were forty-five in which the diagnosis of malignant disease was made by clinical examination before operation. Twenty-eight of these forty-five patients are now dead; seven have a recurrence of the growth and four were not traced. Only six patients of the forty-five whose condition was diagnosed as malignant disease when they were first examined are now alive and well, and in three of these cases only six months or less have elapsed since operation, while the other three patients have lived five years or more since the operation. Only three of the forty-five whose cases were clinically diagnosed as malignant thyroid disease on the first examination are known to be alive after more than five years.

In eleven patients in this series, the presence of malignant disease was not suspected either on clinical examination or at operation and was made only by the pathologist. Three of these are dead, one was not traced and seven are living and well. Four of the seven were operated

on within a year or less, one has lived two years since operation and two have gone four years without further trouble.

The clinical features of malignant thyroid disease when it is well advanced, are distinctive. In many instances the history alone is suggestive of the condition. In twenty-five of our cases, the patient stated that a goiter which had been present for some time became rapidly larger, and in eleven other cases a previous goiter had shown a gradual increase in size. Hoarseness was noted by seventeen patients, either constantly or in repeated attacks. A sense of pressure and constriction in the neck was the presenting symptom in twelve instances, and difficulty in breathing and swallowing was frequently noted. In only three cases had there been attacks of choking, and in but one case was local tenderness a prominent feature of the history. Increasing size of a goiter and local pressure symptoms may therefore be considered the important points in the clinical history that are suggestive of cancer of the thyroid.

It has been our experience that there are not any dependable symptoms or physical signs to indicate the need of radical treatment in early malignant disease of the thyroid. By the time the so-called classic symptoms of malignant disease have developed, the opportunity for successful surgical removal of the tumor is long past.

The most typical symptom on examination in a case of thyroid malignant disease is the firmness or hardness of the goiter. In thirty-four patients in this series, this was especially noted. In many cases, furthermore, the surface or contour of the goiter is quite irregular and feels distinctly nodular. The nodules do not resemble the soft colloid adenomatous swellings seen so commonly in endemic goiter, but are rather hard and firmly attached portions of a centrally malignant tumor. In many instances the malignant condition involves a large part of the gland, and the thyroid is found firmly attached to the adjacent structures in the neck. Here, no one isolated portion of the thyroid gland can be detected on palpation as the point of origin of the growth, but the general resistance and fixation of all the structures give definite evidence of the presence of a malignant condition.

In advanced cases, the tumor is not confined to the usual contour of the thyroid gland and makes its appearance in locations not usually invaded by thyroid enlargement. Thus, a large nodule may lie behind the lower end of the sternomastoid muscle, though it arises from and is connected with the goiter seen in the front of the neck. We have seen the malignant growth push its way beneath the jugular and the carotid and lie entirely behind the carotid sheath in several instances (figs. 9 and 10).

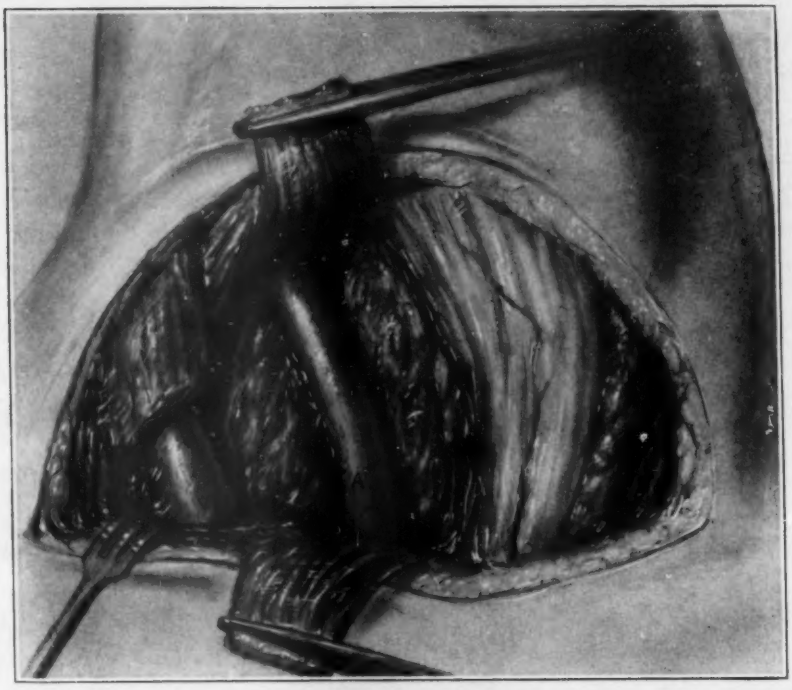

Fig. 9.—Drawing made at operation on a malignant tumor of the right lobe of the thyroid gland. The prethyroid muscles have been cut, and the sternomastoid has also been partially divided. The carotid artery (*A*) and the internal jugular vein (*V*) were displaced anteriorly by the extension of the tumor behind them.

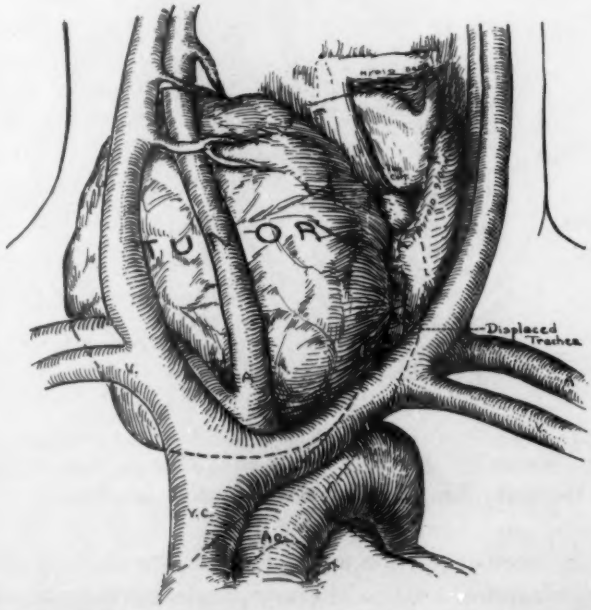

Fig. 10.—Schematic drawing of the tumor shown in figure 9. The distortion of the great vessels of the neck and rotation of the trachea and the larynx by the pressure of the growth should be noted. Failure to recognize that this type of tumor may grow behind the large vessels of the neck may give rise to serious injury to these vessels when the neck is opened. *V. C.* is the vena cava and *Ao.*, aorta.

METASTASES

In many cases, examination will disclose evidence of metastases or of probable metastases of the growth. The most common evidence of this is found in the presence of enlarged lymph nodes in the vicinity of the original tumor. In thirteen of our patients the cervical lymph nodes

Fig. 11.—Drawing of a specimen from the trachea and esophagus in a case of malignant disease of the thyroid. The extension of the tumor tissue into the trachea and the involvement of the esophagus should be noted.

were seen to be enlarged, the most common site of these glands being in the anterior cervical nodes. The supraclavicular and posterior cervical glands may also be involved. In one case, the axillary glands on the side involved by the malignant condition were markedly enlarged. It

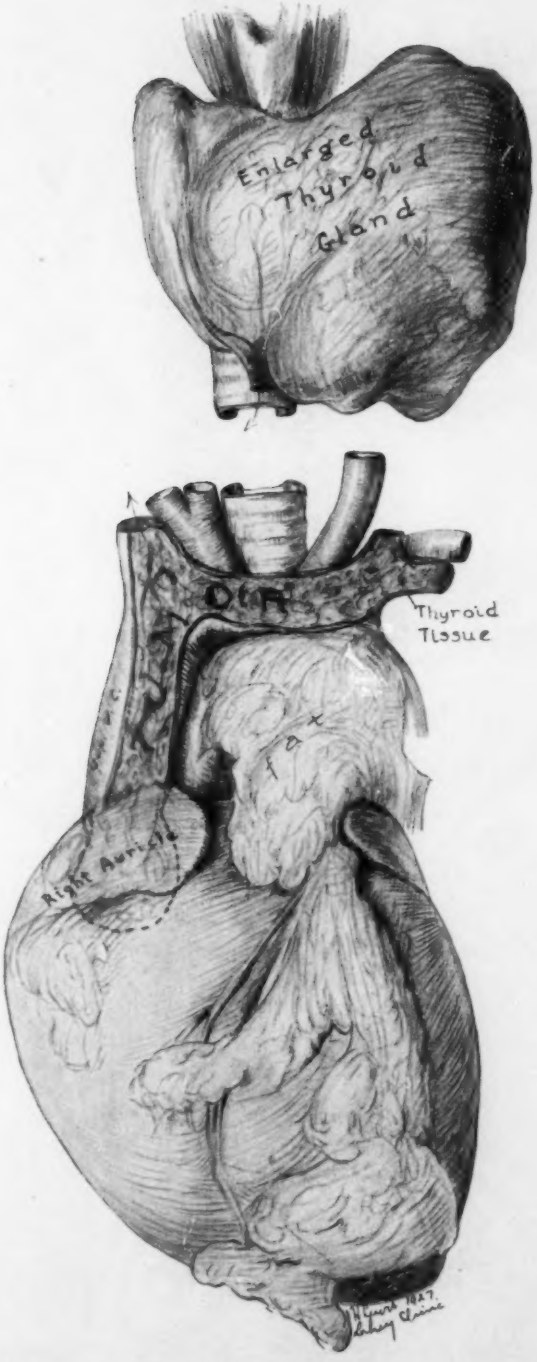

Fig. 12.—Malignant tumor of the thyroid gland with invasion of blood vessels and growth through veins into the superior vena cava and right auricle of the heart. On clinical examination, the tumor of the thyroid was not suggestive of a malignant condition.

was thought that the tumor had involved the axilla in this case, not only by extension along the lymphatics, but by growth in the subclavian and axillary veins. In eight cases the growth had extended into the mediastinum, as a rule by direct extension downward from the neck. In three cases, numerous nodules were present in the lungs. These were so extensive in one case that marked dyspnea and orthopnea resulted from the large amount of lung tissue involved. The trachea and the esophagus

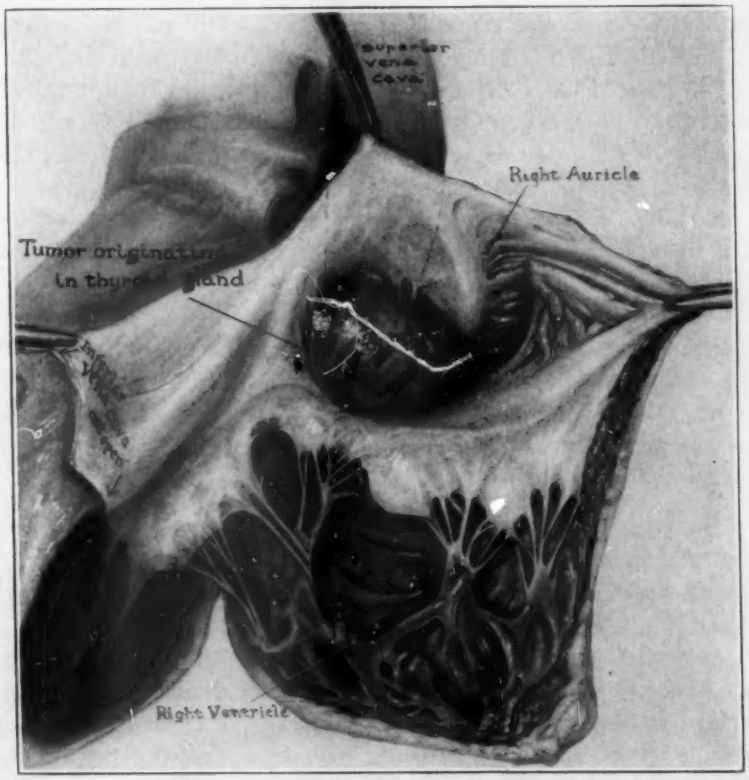

Fig. 13.—Drawing of the right side of the heart in the case shown in figure 12. The large tumor projecting into the right auricle through the vena cava should be noted.

may be invaded by direct extension of the growth (fig. 11), and likewise the muscles adjacent to the gland may be directly involved in the growth. Metastases to bone have been seen in only one case in which numerous nodules were found in the ribs. In another case (fig. 12) the tumor invaded the superior vena cava and grew downward until it projected as a definite nodule nearly an inch in diameter in the right auricle of the heart (fig. 13).

CONDITION FOUND AT OPERATION

The condition found at operation in the frankly malignant thyroid tumor is quite typical. The most suggestive single manifestation is the loss of the various planes of cleavage as one reaches the gland. The sternothyroid muscle, which ordinarily is separated from the thyroid gland by loose cellular tissue, is densely adherent and in many cases cannot be readily freed from the tumor. In many cases the regular contour of the thyroid gland is lost, and the tumor tissue is found invading the adjacent muscle tissue. In certain cases it may be possible to note thrombosed veins lying over the surface of the gland. Inside malignant thyroid lobes we have twice found thick yellowish fluid which resembled pus in an abscess cavity and which gave rise to the erroneous impression that thyroiditis with formation of an abscess was present. In certain cases, the surface of the gland may lose its shiny appearance and become pale red or even milky white. In these cases edema is present in both the thyroid tissue and the overlying structures, and it is often impossible to decide whether the condition is due to malignant disease or to the pressure from a recent hemorrhage in a large adenoma. At operation, the differentiation of Riedel's struma or severe thyroiditis from a diffuse malignant condition involving a lobe or the entire thyroid gland may be impossible. In thyroiditis, however, the various tissue planes are generally well preserved. In well advanced malignant disease, these planes are lost.

TREATMENT

The surgical measures that can be employed in the treatment for malignant disease of the thyroid are not numerous. In many frankly inoperable cases, it has been our custom to remove a small specimen for histologic study. A biopsy is of value first in settling beyond any reasonable doubt the diagnosis and, second, it gives definite evidence as to the type of tumor with which one is dealing. The latter information is of value in estimating what response may be expected from roentgen or other therapy and what the probable duration of life may be. These advantages must be balanced against the fact that a biopsy is not without some danger, since we have had a patient develop fatal pneumonia directly after the operation and, furthermore, we have gained the impression in certain cases that the rate of growth of the tumor was distinctly accelerated after the removal of a specimen.

In many cases, the malignant portion of the thyroid will be found confined to a single adenoma or to part of one lobe of the thyroid gland. Here the operation has been limited to the removal of the adenoma alone or to the complete removal of the involved thyroid lobe. When the malignant portion appears to be confined within the capsule of the adenoma, the wide removal of the adenoma and much of the thyroid lobe in which it lies is valuable. We have never felt that the radical removal of

the entire thyroid gland in extensive carcinoma was indicated, since it is highly improbable that it will remove all the malignant tissue unless the operation at the same time is so extensive that the parathyroid glands are removed, in which case fatal tetany must ensue.

In certain patients with rapidly growing cancer of the thyroid gland respiratory obstruction becomes such a prominent symptom that active measures must be undertaken to relieve it. The possible procedures are removal of part of the obstructing tumor, decompression of the thyroid and tracheotomy. When a portion of the thyroid tumor, especially that portion lying on the trachea, can be removed, marked temporary relief of respiratory obstruction may be obtained. Similar though less marked relief is gained by simply cutting the prethyroid muscles, as recommended by Dr. Crile. While these procedures give a little relief, the benefits derived in our experience have been so fleeting that one really wonders if they are worth while. Tracheotomy, when it can be carefully performed as an operation of election, gives tremendous immediate relief to the patient and is of more lasting benefit, frequently preventing further serious choking spells during the patient's short remaining existence. Because of the extension of the malignant condition toward or into the superior mediastinum, tracheotomy may be impossible unless it is preceded by the removal of thyroid tissue from the front of the trachea.

We have had no experience with radium in the treatment for malignant conditions of the thyroid gland. Dr. Martin Tinker of Buffalo, N. Y., and Dr. Pemberton of Rochester, Minn., have found radium of definite value in these cases and recommended its use at the time of operation and postoperatively. We have found roentgen treatment for cancer of the thyroid valuable in certain cases, notably the malignant adenoma group. Of this group, the papillary adenomas have apparently been more susceptible to roentgen treatment than the fetal adenomas. This treatment has not checked in any remarkable degree the course of the disease in the remaining histologic groups as we have studied them. It is, of course, impossible to state what would have happened in any given case without the addition of roentgen therapy after operation. However, we have the strong impression that roentgen treatment retards the rate of growth and delays the appearance of recurrences in malignant adenomas of the thyroid but is not of benefit in squamous cell, giant cell and small cell carcinoma of the thyroid gland.

SUMMARY AND CONCLUSIONS

1. The incidence of cancer of the thyroid among the cases of goiter in the Lahey Clinic is 1.68 per cent.

2. There were 6.4 times as many women as men who had malignant disease of the thyroid.

3. The greatest incidence of the disease was between the ages of 50 and 65. Cases occurred in persons aged 20 and 82.

4. An adenomatous goiter preceded the malignant disease in 94.4 per cent of the cases studied.

5. Histologically, cancer of the thyroid may be divided into four chief groups: (1) malignant adenoma; (2) squamous cell carcinoma; (3) giant cell carcinoma, and (4) small cell carcinoma. Cases in each of these groups have a marked similarity in their clinical history and course.

6. Knowledge of the histologic group of a cancer of the thyroid gives valuable information as to (*a*) the chances of recovery of the patient or (*b*) the probable duration of life and (*c*) the advisability of roentgen treatments.

7. When cancer of the thyroid can be diagnosed clinically, the chances of cure are small. Lowering the mortality of thyroid malignant disease must at present depend on the removal of the premalignant adenoma.

8. Surgical removal and postoperative roentgen treatment give some good results in the first histologic group (malignant adenoma). Treatment has not been of any real value in the other types of malignant disease of the thyroid.

OBSTRUCTIVE HYDRO-URETERAL ANGULARITY WITH HYDRONEPHROSIS IN CHILDREN

SURGICAL TREATMENT *

FRANK HINMAN, M.D.
SAN FRANCISCO

The general policy of making a complete urologic examination of children suffering with chronic pyuria has brought to light an interesting and as yet poorly classified group of pathologic conditions. In the majority of instances, an obstruction at some portion of the primary tract

The Direct Pathologic Effects of Obstruction

	I. Local Changes (Obstruction)	II. General (Infection)	III. Functional
In the urethra	Extravasation of urine Abscesses Urinary fistulas	Urinary sepsis, whether ascending or descending, localized or progressive Urethritis	Disturbances of urination: frequency burning, dribbling, incontinence, retention
In the bladder	Trabeculation Cellule formation Diverticulum Hypertrophy of interureteral ridge Dilatation Rupture	Cystitis	Renal insufficiency
In the ureter	Hydro-ureter Ureteral hypertrophy and angularity Ureteral diverticulum	Ureteritis	Uremia
In the kidney	Hydronephrotic atrophy Secondary atrophy	Pyelitis Pyelonephrosis Infected hydronephrosis Pyonephrosis	

has been the primary factor in the chronicity of the pyuria. The obstructive syndrome is probably the same in children as in adults. Although pain is not so prominent, the sequence of changes of obstruction-pain-infection obtains, and these effects are due to various causes of back pressure, stasis or back-flow, just as in adults. Infection is always imminent in the presence of any of these, and the clinical effects in their relation to the pathologic changes are diagrammatically represented in the foregoing outline.

The nature of the causes that are known to produce these local, general and functional effects is complex and often difficult of recognition. They are generally grouped as congenital or acquired in nature, and it is a common practice to speak of the pathologic effects of back-

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pressure as congenital when, strictly speaking, the cause only is congenital. The effects, such as diverticulum, hypertrophy of the interureteral ridge, hydro-ureter and hydronephrosis, are acquired. Naturally, the congenital causes would be expected to be more frequent in the obstructive conditions of childhood than the acquired ones and, although the discussion of causes is not my object in this communication, they are listed herewith in order to emphazise this point of departure and to define more clearly the condition of obstructive ureteral hypertrophy and regularity.

Causes of Obstruction

A. CONGENITAL CAUSES

I. Of the Lower Tract:

Long tight foreskin
Congenital stricture of the urethral meatus
Posterior urethral valves in boys (hypertrophied verumontanum)
Urethrocele or diverticulum
Abnormal opening and fistulas (hypospadias, epispadias, exstrophy)
Contracture of the vesical neck

II. Of the Upper Tract:

1. Urethral Anomalies:

(a) Of Form:

(1) Valves or folds (usually associated with other conditions and therefore difficult to say whether or not they are all acquired or secondary causes of obstruction)

(2) Megalo-ureter (congenital defect of innervation or of muscle development—persistence of fetal type ureter into infancy)

(b) Of Division:

(1) Double or triple
(2) Diverticulum

(c) Of Termination:

(1) In the bladder wall, producing kink,
valve,
pouch or diverticulum,
ureterovesical cyst

(2) Abnormal or blind openings—in urethra, intestines or accessory sex ducts; (male) seminal vesicles, ejaculatory ducts, etc.; (female) vagina, uterus, etc.

2. Pyelorenal anomalies:

(a) Of form and number (double pelvis, horse-shoe kidney or solitary lump kidney)

(b) Of size

(c) Of position (incomplete ascent)

(d) Of vascularization (aberrant vessels or bands, producing kink or valve, the commonest cause of so-called congenital or idiopathic hydronephrosis)

(See II-1-b 4 in section B of this table)

B. ACQUIRED CAUSES

I. Of the Lower Tract

1. Urethral:

Phimosis
Stricture
Stone
Tumor
Diverticulum

2. Urethrovesical:

Prostatism (such as hyperplasia, cancer, median bar and contracture of the vesical neck)
Prostatic cysts
Urethrovesicle polyps

3. Vesical:

Tumor
Stone
Diverticulum
Hypertrophy of the interureteral ridge
(The last two usually secondary)

II. Of the Upper Tract

1. Ureteral:

a. Local:

Stone and foreign bodies

b. General:

(1) Stricture and stenosis (1, focal [Hunner] or 2, local [as in pelvic inflammatory, vesiculitis], 3, traumatic)
(2) Kinks or angularity (from bands, blood vessels, inflammatory or postoperative adhesions)
(3) Pressure from outside (pregnancy, pelvic tumors, etc.)
(4) Ureteropelvic conditions with ptosis (valve, kink, fold, stricture, ureter is fixed and does not follow movements of kidney)
(Closely related to anomalies of vascularization, A-II-2-d)
(5) Ureterovesical conditions (stricture of intramural inflammation)
(6) Diverticulum (secondary)
(7) Ureteral hypertrophy and angularity (secondary)

2. Pelvic (ureteropelvic):

Stone
Tumor
Parasites

3. Renal (ureteropelvic):

Stone
Tumor
Ptosis with kink or angulation, related to 1-b-4
(From aberrant bands, adhesions or blood vessels)

III. Dilatations without evidence of obstruction
(Functional disturbances, adynamic conditions such as might be the result of nerve disturbance. Tabes, paresis, etc.)

If one were to indicate in the list shown in the table the most frequent congenital causes of obstruction, abnormalities of the posterior urethra and the bladder neck in the lower tract and the anomalies of division and of termination of the ureter and the anomalies of vascularization of the upper tract would probably be selected. Analysis of the acquired causes is a much more difficult matter. In men prostatism is common, but changes in the lower tract dominate the clinical picture. Acquired conditions of the upper tract in men, women and children form a confused, unclassified group about which few urologists agree. The relative importance and significance of ureteral stricture, ureteral kinks and renal mobility are undetermined and will probably remain so until methods of study and interpretation have become standardized. But it is not my purpose to enter this controversy here, and I have outlined it in order to present more clearly a certain type of obstruction that follows various of the foregoing conditions, both congenital and acquired, mechanical and dynamic or primary and secondary.

This special condition is best defined by the term obstructive hydroureteral angularity. It is frequently encountered in children, and seems to me to be secondary and not primary. It is often difficult and sometimes impossible definitely to determine a primary cause of obstruction when marked back-pressure changes have occurred, even in adults it has long been recognized that certain of these back-pressure changes can of themselves produce obstruction. Diverticulum of the bladder and hypertrophies of the interureteral ridge, although secondary to an obstructive condition, can so develop as to produce of themselves marked obstruction to the urinary outflow. In my opinion, the following group of cases illustrates a similar condition of secondary obstruction but one that occurs in the upper urinary tract, even though it has been impossible to determine definitely the primary obstructive condition in a few of the cases.

If my assumption is correct, the condition of posterior urethral valves in boys would be ideal for the production of obstructive hydroureteral angularity. Relief of the urethral obstruction would not be of permanent benefit in those patients who have developed this secondary ureteral condition. With this point in mind, I have recently analyzed my cases.

ANALYSIS OF CASES

Of the twelve patients seen to date with posterior urethral valves, four are dead of uremia and two have disappeared from observation. In the six known to be alive the urethral valves were destroyed—in one, by cystotomy; in five, by fulguration per urethram; in one, six years; in one, four years; in one, three years; in two, two years, and in one, one year before this paper was written. Of the patients who were

not followed, one was treated by fulguration five years and the other two and a half years before the present admission. The condition of the upper urinary tract in these two patients was not studied at the time of the first admission, and they failed to return for investigation. Urethral valves, however, were definite and every effort is being made to get these patients back for further study. The four patients who died all had huge bilateral hydro-ureters and hydronephroses and died of urinary sepsis and uremia. One,[1] aged 18, was uremic on admission and died in the hospital under treatment with a retention catheter. One,[2] aged 6½, "never regained his health after leaving the hospital in 1917" (valves destroyed), though "he went through grammar school and was in his sophomore class in High School when we lost him on March 24, 1927," according to a note signed by the father. He was 16 when he died. The third, aged 21, died twenty-seven days after perineal resection of the valves which was performed after thirty-five days of catheter preparation during which time renal function had improved from no dye excreted in two hours to an output of 21 per cent. The nonprotein nitrogen climbed to 230.7 mg. per hundred cubic centimeters a few days before death. The fourth patient, aged 21 months, died a few days after unilateral nephrostomy. The urinary tract, which was removed intact after death, illustrates the condition I am attempting to describe of bilateral obstructive hydro-ureteral angularity (fig. 1). The upper right tract has shrunken considerably following the relief of back-pressure by nephrostomy, but the elongation and tortuosity of both ureters are remarkable, and only a thin shell of renal tissue remains in each hydronephrotic sac.

Four of the remaining six patients who are alive show bilateral obstructive hydro-ureteral angularity of varying degree. Three of these cases are reported later in the paper in connection with surgical treatment (cases 4, 5 and 6). In two of the six the changes in the upper tract do not seem to have advanced to a degree sufficient to produce secondary urinary obstruction, but complete studies were not made in either. One patient was only 2 years old on admission, Jan. 14, 1926, and had a total phthalein output on January 16 of: first hour, in 60 cc. of urine, a trace; second hour, in 100 cc., 21 per cent; total, 21 per cent. Three obstructing folds of mucous membrane in the posterior urethra were destroyed by fulguration. One year later the phthalein output was more than 50 per cent in two hours, and the child, now aged 3 years, was clinically well and the urine normal. But, in view of subsequent experience, I believe that this child should be reexamined.

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1. Case 6 in Hinman, Frank: Congenital Valvular Obstruction of the Posterior Urethra, J. Urol. **14**:71 and 83, 1925.

2. Case 1 in Hinman (footnote 1).

The other boy is 11 years old and was seen only three months before this paper was written. The valves have been destroyed but the enuresis, which led to their discovery, still persists and the boy is returning soon for further investigation. The condition of the upper tract in the other case, which is believed to be one of bilateral obstructive hydro-ureteral angularity, is shown in figures 2 and 3. This boy was 4 years old in

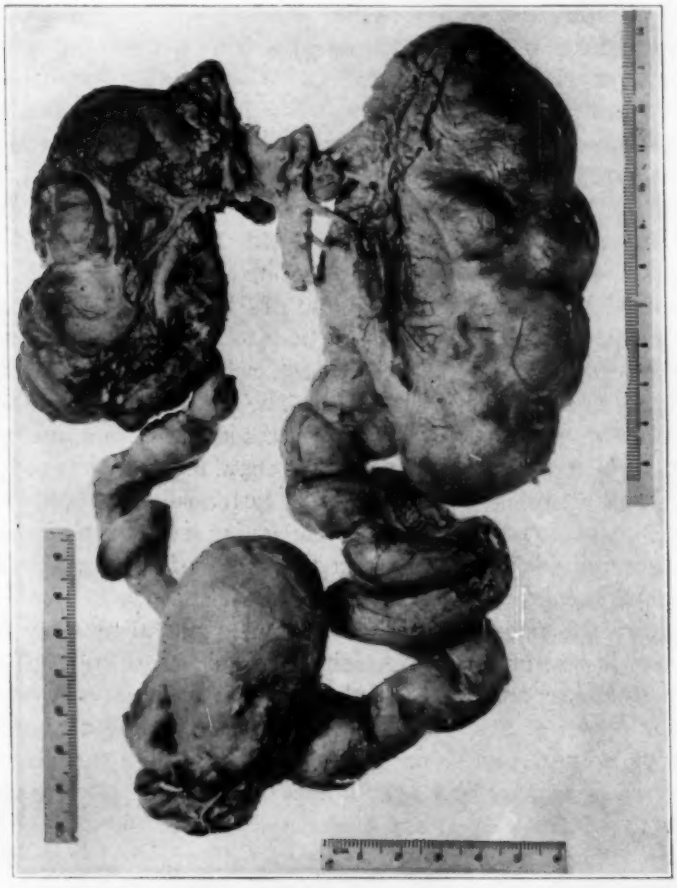

Fig. 1.—Kidneys, ureters and bladder with the posterior urethra removed en masse at autopsy. The right side was shrunken on account of nephrostomy drainage. The left ureter is from 2.5 to 3 cm. in diameter in places.

June, 1922, when these pictures were made. The valves were destroyed by fulguration, July 11, 1922. His mother wrote, in February, 1928, that "he is growing fast, is somewhat above normal weight, general health is very good" but "a very little albumin and a few pus cells in the urine" were reported by the local physician who was giving him "hexylresorcinol, one capsule three times a day after meals." On

Dec. 27, 1923, this boy came back for observation. There were from 8 to 10 pus cells in a high dry field, but the total phthalein output was low, only a faint trace being returned in fifty minutes and after four hours only 10 per cent. Residual urine was not found but reexamination of the upper tract showed an enormous left hydro-ureter and hydronephrosis. The right was not shown in the cystogram. If the results of the surgical treatment in the other three cases reported below are at all successful, this boy should be operated on also as he cannot survive long under the present conditions. He has bilateral obstructive hydro-ureteral angularity.

Fig. 2.—Posterior urethral valves in a boy, aged 4 years. This case was described by Hinman, Frank: Congenital Valvular Obstruction of the Posterior Urethra, J. Urol. **14**:76, 1925.

So far, the assumption that hydro-ureteral angularity is secondary may withstand criticism because all of the foregoing cases show a definite primary obstruction of the lower urinary tract. However, similar and bilateral conditions have been observed in girls, in some of whom it has been impossible definitely to locate an obstruction of the lower tract.

A girl, aged 3 years, was admitted to the hospital in October, 1926, complaining of periodic attacks of fever and pronounced pyuria, but no urinary symptoms. Plain roentgenograms were negative, and the total phenolsulphonphthalein output was 43 per cent for the first hour and 10 per cent for the second hour. The two ureteropyelograms illustrated in figures 4 and 5 show a condition of hypertrophied

tortuous ureters similar to that described in boys. Attempts to get this patient back so as to find out what condition exists at the present time have failed. The cystoscopic study did not show an obstructive condition at the bladder neck or in the urethra. One may have been present in the lower ureters.

The condition in another infant, referred by Dr. Lucas, is both interesting and suggestive in this connection. This patient, a girl, was 15 months old and had been suffering for about six weeks with periodic attacks of vomiting associated with high fever. For the last three months the mother had noticed that the urine

Fig. 3.—Ureterogram obtained on left side in the boy referred to in figure 2.

had a strong odor, and examination of the urine by Dr. Lucas revealed a pronounced pyuria. Urologic study showed a rather dilated bladder with a capacity of about 500 cc. The ureteral orifices appeared dilated, the one on the right in particular being the shape of a golf-hole. The left seemed to be somewhat redundant with a mucous flap, although there was no obstruction to the passage of catheters, and ureteropyelography on subsequent days showed somewhat dilated ureters and a mild degree of hydronephrosis on both sides. These kidneys were washed twice. The infant was last seen on March 8, 1927, when a no. 10 bulb was easily passed to the pelvis of each kidney. Obstruction of the lower tract was not found.

It will be an interesting study to see whether in this case the ureteral and pelvic changes will be progressive. When this child reaches the age of 5 to 11, will the condition of the upper urinary tract be the same as in most of the other cases? It is, of course, possible that the ureteral dilatation will have relieved somewhat the obstructions present but, as there was no particular difficulty in passing the bulbs, this is unlikely.

Fig. 4.—Ureterogram of girl, aged 3 years, showing a markedly dilated, angular ureter without as yet much hydronephrotic atrophy, a condition more nearly analogous to what is usually refered to as megalo-ureter. The condition of the opposite kidney is illustrated in figure 5.

The defect would seem to be from above downward rather than from below upward.

That the condition may be unilateral but acquired and not congenital would seem to be shown by the condition found in a girl, aged 11 years, referred because of a long standing pyuria and recurrent attacks of fever and pain in the right side. She had been advised, and probably

rightly so, in another city where urologic study had been made, to have a right nephrectomy performed. Through a no. 21 F cystoscope a large, balloon-like mass about 7 cm. in diameter projecting from the right ureteral orifice was seen. The surface of this ureterocele, or ureterovesical cyst, was easily indented by the tip of the catheter, but the ureteral orifice was not seen or found by probing. On overdistention of the bladder, however, the cyst collapsed completely and dis-

Fig. 5.—Ureteropyelogram on the left side of the kidneys of the patient in figure 4.

closed a large, dilated orifice; when attempts were made to catheterize this the catheter would coil up in the cavity. The wall of the cyst was destroyed by fulguration, and then the catheter could easily be passed up the right ureter. The ureteropyelogram then taken is reproduced in figure 6 and shows marked angularity and dilatation in both the upper and the lower thirds of the ureter, in every way similar to the ureteral changes following partial obstruction in the posterior urethra already described. The patient was advised that a left ureteropyelogram should be taken and that, if this side was normal, she should wait a short

time to see if removal of the lower ureteral obstruction caused repair of the hydronephrosis. If not, the degree of improvement in function with a retained ureteral catheter should be tested as an indication of whether nephrectomy should be performed or an attempt made to repair the hypertrophied, dilated and angular ureter. If the left kidney is found to be abnormal, conservation would be even more indicated. In spite of

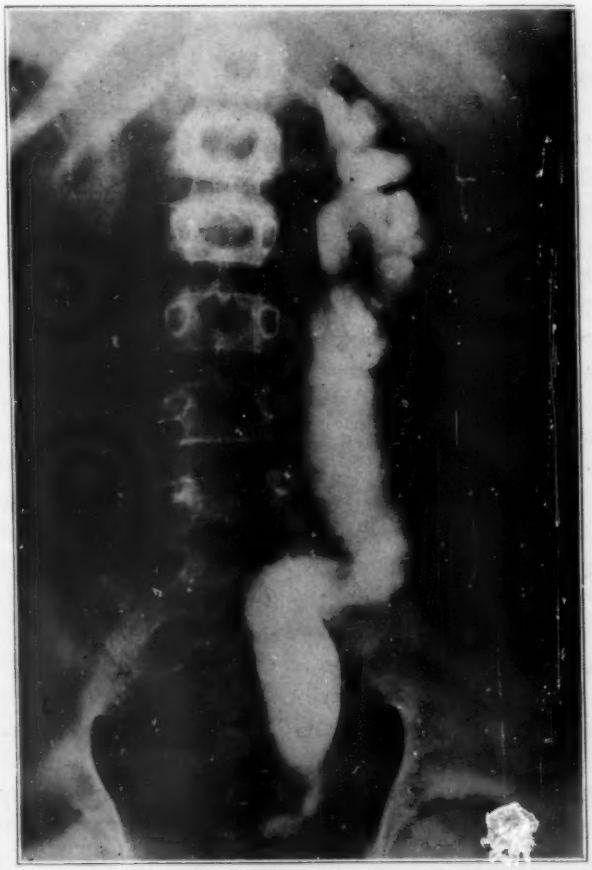

Fig. 6.—Hydro-ureteral angularity resulting from obstruction due to ureterovesical cyst.

the low function, as evidenced by absence of phenolsulphonphthalein on the right side at the time of the first ureteral catheterization, experience has shown that a most remarkable improvement in function follows good drainage even when the remnant of renal tissue is small. The advisability of conservative surgery would be well shown by the degree of improvement in function following the use of a retention catheter for a few days. This patient returned home, several thousand miles

away, had another attack of fever and pain and a right nephrectomy was performed. This, no doubt, was the simplest and safest procedure in view of the probabilities that the remaining kidney is normal, but the possibilities of conservation and repair of such kidneys in children should not be overlooked.

DEFINITION AND DIAGNOSIS

Obstructive hydro-ureteral angularity is an acquired condition that follows a partial obstruction, the cause of which may be congenital or acquired and is always accompanied by hydronephrosis. It is secondary to the primary obstruction just as much as the hydronephrosis is secondary. Hydronephrotic atrophy is the pathologic effect of obstruction on the pyelorenal structures, hydro-ureteral angularity, the pathologic effect of the obstruction on the ureter. The primary obstruction producing it is always in the lower tract or the lower ureter. It is to be at once distinguished from cases of congenital or other types of atony of the ureter in which there is marked relaxation and dilatation but without hypertrophy of the wall, tortuosity or pronounced elongation, a type of case which has been recognized clinically as megalo-ureter or ureteral atony. The condition also is to be differentiated from cases of hydronephrosis in which the obstruction is definitely in the upper ureter or at the ureteropelvic juncture, the common cause of which is an aberrant blood vessel. The condition is also to be distinguished from the common type of hydro-ureter seen in adults of which there is no marked angularity, although adults can develop an obstructive hypertrophy and angularity. The distinction lies in the twists and corkscrew deformities that have developed as a result of peristaltic overactivity. A certain prolonged but mild type of obstruction seems required. The ureteral wall hypertrophies and elongates as well as dilates, and the angularity produced forms kinks and bends that become bound together by adhesions and collateral blood vessels. Because of the fixation of the angularity, urinary obstruction results. The angularity is most marked at the lower and upper portions but, as well shown in the specimen of the advanced case illustrated in figure 1, may involve the whole ureter. When the condition exists, removal of the primary obstruction will not give permanent relief of the progressive hydronephrosis.

The condition is diagnosed by cystography and ureteropyelography. Cystography is usually successful in demonstrating the changes in the upper tract in all cases showing obstructions of the lower tract. Ureteral catheterization of the angular, corkscrew hydro-ureters is often impossible, the catheter coiling up in a dilatation below some lower angle, but even from here the dilated ureter can frequently be filled for

ureterography. The coiled position of the catheter as seen in the roentgenogram is a good indication of the ureteral condition even when the pelvis cannot be reached. But the very nature of the condition precludes the possibility of an obstruction from below once the primary factor of obstruction is passed, so that I have never failed, on the second trial, at least, to outline the ureter and pelvis even with the catheter coiled in the lowest sacculation.

COMMENT

The confusing feature in the classification of this condition as secondary to some other obstructive condition is its occurrence in girls in whom obstructions of the lower tract cannot be found. In a few cases there is slight evidence of some hypertrophy of the bladder muscle, indicating that there might have been some mild obstruction of the lower tract. Pyuria, frequency, enuresis or dribbling are commonly found in the history, but it is hard to conceive how chronic irritation causing increased frequency brings about such marked changes in the upper tract. It is more probable that the condition has some definite relationship to ureteral development and growth as it is most marked in the very young, although one finds similar back-pressure changes in adults. Back-pressure ureteral changes from acquired obstruction, as seen in adults, do not as a rule show the same degree of redundancy and angularity that develops in the young. The explanation of the condition as secondary to obstruction is plausible when it is found in association with those cases which present posterior urethral valves, and with other cases showing definite obstructive conditions at the ureterovesical juncture. It is possible that some of these hypertrophied hydroureters with angularity found in girls without definite obstructions of the lower tract were partially obstructed because of the abnormal manner in which the ureter opened into the bladder, or the abnormality may have started with a temporary obstruction due to a uric acid calculus. Beer has emphasized the frequency of this condition in childhood. An analogous condition is often observed in experimental animals whenever a partial obstruction of the lower ureter has been produced. I recall an experiment in which a rubber band had been so placed about the lower ureter as partially to obstruct it. But one year later, when the ureter was explored with the dog anesthetized, it was seen to be undergoing marked peristalsis and had so elongated itself as to be kinked and angular just as the hydro-ureters of the children whose cases have been reported previously. These tortuous angularities, however, had not been bound together with the firm adhesions and collateral blood supply as in the foregoing clinical cases, and retransplantation to the bladder effected repair of the hydronephrosis.

A retention ureteral catheter, when it can be properly placed, is the most valuable method of determining the advisability of subjecting these patients to operation. Improvement in renal function on retained ureteral catheterization gives reliable indication of the improvement that may be expected when the urinary outlet is made free surgically.

It must be kept in mind in connection with consideration of treatment that renal repair in children is a different and much more hopeful problem than in adults. Pathologists have shown that new formation of glomerular and tubular units continues after birth up to the seventh to the tenth years. Probably even with proper stimulation there would not be a greater number of glomerulotubular units formed than occurs normally, but some recent experimental work on compensatory hypertrophy in rats shows clearly that the rate of development of these new, forming secretory units and the degree of hypertrophy attained are markedly accelerated in the young animals as compared to the adults.[3] Repair of an infant's kidney might be greater and more permanent and efficient than from a similar pathologic condition in an adult. But the obstruction must be completely and permanently removed in order to secure this.

Before the surgical treatment is discussed, it may be of interest to present briefly in chronologic order the cases in which relief of this type of obstruction of the upper tract was effected by surgical intervention. Mistakes are often instructive; at least they have led me to consider the surgical procedure adopted in the last four cases and presented in detail in the summary as the most suitable for obstructive hydro-ureteral angularity.

REPORT OF CASES IN WHICH OPERATION WAS PERFORMED FOR OBSTRUCTIVE HYDRO-URETERAL ANGULARITY

CASE 1.[4]—D. V., a girl, aged 6 years, was admitted to the hospital in 1918 with the complaint of persistent pyuria for three years with marked frequency and burning on urination for the past three months. The child was normally developed but poorly nourished. The urine was cloudy, foul smelling, loaded with pus and contained a few casts. Plain roentgenograms were negative. The phenolsulphonphthalein output on admission was less than 10 per cent in two hours. Cystoscopic examination showed pronounced cystitis with bilateral hydro-ureters and hydronephroses. On account of the advanced nature of the trouble it was thought that nothing could be done, and after a few pelvic lavages the child was sent home. She reentered the hospital in April, 1920, and had done fairly well up to the last three weeks when the frequency, burning and pain on urination recurred worse than ever. Reexamination revealed the situation little changed. Total renal function

3. Hinman, Frank: Renal Counterbalance, Arch. Surg. **12**:1105 (June) 1926.

4. Reported as case 7 in Hinman, Frank: Renal Counterbalance: An Experimental and Clinical Study with Reference to the Significance of Disuse Atrophy, Tr. Am. A. G. U. Surg. **15**:241, 1922.

of phenolsulphonphthalein at this time was 14 per cent in two hours. Ureteral retention catheters were left in for several days, and there was a marked improvement in kidney function with the catheters in place. On May 8, 1920, the phenolsulphonphthalein test showed, by way of the left catheter, a return of: first hour, 10 per cent; second hour, 20 per cent; whereas, by way of the bladder from the right kidney, only a faint trace during the first hour and 3 per cent during the second hour were obtained. Previous to the use of the retention catheter, only a slight trace of phthalein had ever been obtained from either kidney. At operation,

Fig. 7 (case 1).—Cystogram with left ureteral catheter in place taken just a few weeks before the last operation, at which from 8 to 9 cm. of each ureter was resected at its lower end and the open end reimplanted into the bladder.

in May, 1920, a left ureteropyeloplasty was performed. The upper ureter was freed of adhesions and straightened of its angularities but even then, when left to itself, would coil back into nearly its original position, producing an apparent valvelike kink at the ureteropelvic juncture. A Heinecke-Mikulicz procedure was performed at this point, the same as in pyloroplasty, with the idea of relieving this obstructing angle. Two weeks later the total renal function, as shown by the phenolsulphonphthalein test, was: first hour, 10 per cent, and second hour, 15 per cent. Three weeks later the output during the first hour was 20 per cent and during the second hour, 15 per cent. On June 2, one month later, with the ureteral catheters in place,

the return during the first hour was 30 per cent and the second hour, 30 per cent, a satisfactory total of 60 per cent. On June 8, a similar operative procedure was performed on the right side. On July 3, the total phenolsulphonphthalein output for the first hour was 20 per cent and for the second hour, 10 per cent, ureteral catheters not having been used for a week or so. The patient was discharged on July 11.

The patient reentered the hospital on Aug. 9, 1920. The general condition had improved; she had gained 7 pounds (3.2 Kg.); the wounds in both kidneys had completely healed, but between July 31 and August 5 there were fever, nausea and vomiting, and the amount of urine decreased and became quite cloudy, with a foul odor. This condition lasted five days and then cleared up completely. On August 9, the total phenolsulphonphthalein output for the first hour was 25 per cent and for the second hour, 25 per cent. The urine still showed numerous pus cells and many rods. During this month, a number of pelvic lavages were given.

On Dec. 3, 1920, the phenolsulphonphthalein output for the first hour was 20 per cent and for the second hour, 15 per cent. The patient had not been examined subsequent to this date, up to May 17, 1928, a period of almost eight years. She was heard from in April, 1922, and her condition had remained satisfactory; she was gaining, going to school and had not had any further febrile attacks.

Just eight years after the second ureteropyeloplasty, on May 17, 1928, the patient returned to the hospital for observation at my request. She was a healthy looking girl of 16, and had a good color and no particular complaints. Puberty occurred normally, but the urine still showed much pus and the renal function, as shown by phenolsulphonphthalein, was low, only a trace of the dye appearing in two hours. The condition of the upper tract is illustrated in figure 7. As formerly, renal function immediately improved under retention ureteral catheters. The day of entry, the phenolsulphonphthalein output was only 5 per cent in two hours.

Can anything be done at this late date to stop the progressive hydronephrosis and bring about repair? As things are, this girl will never reach her third decade. The plastic operations performed eight years ago have been a failure because of the obstructive conditions below in the angular ureter.

This patient has been operated on again and the operation performed is described later in the paper.

CASE 2.—B. C., a girl, aged 4 years, was brought to the hospital for examination because of a persistent enuresis and marked frequency of urination during the day. The child had made a poor gain in weight for two years and had not gained for the last seven months. The urine was purulent and showed many motile rods. There was a 31 per cent output of phenolsulphonphthalein in two hours. The blood chemistry was normal. Plain roentgenograms were negative. Cystoscopic examination showed a mild trabeculation of the bladder with a capacity of about 100 cc. Obstructive conditions could not be seen at the neck or in the urethra. Both ureteral orifices were slightly relaxed and dilated and easily catheterized. The urine from the right kidney was clear and free from pus and that from the left, cloudy and quite purulent. On Aug. 21, 1926, the relative function showed: right, an output of 25 per cent; left, a faint trace in thirty minutes; on August 24, after the retention ureteral catheter was inserted, the output was: right, 20 per cent and left, 10 per cent in thirty minutes. The ureteropyelograms in this case are shown in figures 8 and 9.

On September 4, on account of a peculiar mucus fold over the left ureteral orifice, which seemed in some way to obstruct its outlet, the orifice was slit open with the fulguration needle and two retention catheters inserted. Marked reaction followed this procedure, and a tender, painful mass developed in the left upper quadrant; this condition was accompanied with vomiting and acidosis. On September 13, a left pyelostomy for drainage was performed, but the pyrexia continued and catheterization now revealed a marked infection of the right kidney. The child's condition was poor.

Fig. 8 (case 2).—Right ureteropyelogram, showing a mild degree of hydroureteral angularity as compared to the opposite side illustrated in figure 9. This picture should be compared with that just recently obtained, figure 10.

On September 18 a right nephrostomy was performed, with only slight improvement. The fever and sepsis persisted, so that on October 26 a left nephrectomy was considered necessary. Following this, there was immediate and marked improvement. On November 8 the temperature was normal; the child was gaining in weight and appeared well and had a total renal output of 67 per cent of phenolsulphonphthalein in two hours. The right nephrostomy opening had closed, and the child left the hospital.

After discharge she had several attacks of pyrexia with nausea and vomiting. In three of these she was so ill that a right retention ureteral catheter had to be placed, improvement always following. For several months she had been gaining, but the urine was still purulent.

On May 12, 1928, she came back for observation. The total phenolsulphonphthalein output was only 8 per cent. The right ureteropyelogram showed definite progression in the degree of hydronephrosis on the remaining right side (fig. 10;

Fig. 9 (case 2).—Left ureteropyelogram, showing advanced degree of hydroureteral angularity with hydronephrosis. This kidney was removed.

compare with fig. 8). It was thought that unless free drainage was established, it would not be many years before renal insufficiency occurred. Because of this conviction, another operation was advised. On June 2, 1928, another operation was performed, which is described later in the paper.

Case 3.—*History.*—F. W., a girl, aged 8 years, was admitted to the hospital for examination and treatment of enuresis. She was an inmate of the California School for the Blind, her eyesight having been lost between the ages of 1 and 2 years following scarlet fever, supposedly because of a staphylococcus infection. The

urine showed a faint trace of albumin and was loaded with pus cells and many motile rods, but no casts. The renal function showed an output of 4 per cent phenolsulphonphthalein in the first hour and of about 3 per cent in the second hour. Plain roentgenograms were negative. The urologic study showed a bilateral infection and dilated corkscrew ureters and hydronephrosis. The loops in the ureter seemed in themselves to produce definite obstruction as shown by the ureteropyelograms of figures 11 and 12.

Fig. 10 (case 2).—Right ureteropyelogram taken just before the last operation, showing the advance of the hydro-ureteral angularity and hydronephrosis of this remaining kidney.

Operation.—On Sept. 24, 1927, left nephrostomy, right nephrostomy and resection with reanastomosis of a portion of the upper right ureter were performed. The left kidney was exposed, revealing a markedly dilated pelvis and pronounced angularity of the hydro-ureter. The twists and bends were bound together by dense adhesions that were freed with considerable difficulty. After the angularities were freed, the ureter seemed to be from 8 to 10 cm. longer than previously, but straightening it out seemed to free it of any obstructive condition and so it was not resected. The patient was turned and a right lumbar incision made, exposing a

markedly lobulated hydronephrotic right kidney. The ureter on this side was more tortuous and dilated than the left, and these tortuous angularities were bound together with adhesions, as had been the case on the opposite side. After the ureter was straightened, it was 12 cm. longer than before, and a portion about 8 cm. in length was resected, the two ends being anastomosed. There was marked collapse of the kidney following emptying of its pelvis, which indicated that not much secreting parenchyma was left. A drainage tube was placed through an opening in the pelvis down the ureter through its point of anatomosis with the

Fig. 11 (case 3).—Right ureteropyelogram, showing the advanced hydro-ureteral angularity of a girl, aged 8. The left side shows much less back-pressure change, as shown in figure 12.

pelvis and another tube passed through the same pyelotomy opening up into the kidney pelvis. A nephrostomy opening was not made. Convalescence was not particularly complicated; the tubes were frequently irrigated, and when the tubes drained well the temperature would remain about normal. There was improvement in the general condition, but on November 7, both the catheters came out; the temperature rose to 40 C. (104 F.), and the urinary output was markedly diminished. On account of the low hemoglobin content and red blood cell count, the patient was given two transfusions with citrate of whole blood on October 29 and on November 16. On account of the definite improvement in the general condition

as long as the catheters drained and the immediate setback with their removal, operative treatment of the lower ureters was considered necessary.

Second Operation.—On Nov. 17, 1927, a midline suprapubic incision was made; the bladder was exposed and the peritoneum dissected back, exposing the right ureter which was found to be markedly dilated and tortuous. There was a pronounced kink or angularity in the lower third about which the ureter was quite markedly fibrous and thickened, giving the impression of a large, round stone in

Fig. 12 (case 3).—Left ureteropyelogram, showing rather marked hydronephrosis but less marked hydro-ureteral angularity.

the ureter. It was found necessary to resect about 4 cm. of this portion of the ureter, reanastomosing the ureteral ends. This was the ureter that had had 8 cm. of its upper portion removed fifty-three days previously. An investigation of the lower left ureter was not attempted on account of the patient's rather poor condition after the foregoing operation was completed. The postoperative course was satisfactory. The suprapubic tubes were removed completely on the tenth day, and the incision had completely healed before the twentieth. The general improvement was much more marked than that following the first operation. The patient's general appearance was good; she had a good appetite, had not had fever for the

last four weeks and voided normally; the urine, however, was still infected. Total renal function as shown by the phenolsulphonphthalein test on Jan. 17, 1928, was 60 per cent. On May 17, 1928, the condition was only fair. The suprapubic fistula periodically opened and drained for a few days. Total renal function was 28 per cent.

The three surgical cases reported were all in girls in whom obstructions of the lower tract were not found, a condition that has been designated by some as congenital atony of the whole urinary tract. The primary factor at the bottom of these progressive changes is not known. It is worthy of note that the degree of change in the right and left upper tract was unequal when these patients were first seen. In one (case 2) the bladder had a small capacity (100 cc.) with mild hypertrophy of its muscle walls, although obstruction was not found. The right kidney showed only a moderate hydronephrosis and it functioned well as compared to the left kidney, but within two years there had been marked advance in the back-pressure changes of this remaining kidney. Whatever the initial lesion or defect that starts the ureteropelvic apparatus to its march of dissolution, when the condition is bilateral it is fatal. But the marked ureteral changes of hypertrophy and angularity are not consistent with atony. They are the well recognized effects of obstruction and can be reproduced in animals by partial ureteral blocks of the lower portion. A satisfactory explanation is wanting, but the marked improvement on catheter drainage is an indication that these cases are not hopeless and that continued efforts to find a means of relief may lead one eventually to stumble on the true explanation of their pathogenesis. The appearance of the bladder neck in the two girls that were operated on as seen suprapubically (cases 1 and 2) is of interest. The iris-like folds of mucous membrane were not recognized cystoscopically. The inequality of the changes on the two sides is also a marked characteristic in the following cases in boys.

CASE 4.—*History.*—B. B., a boy, aged 5½ years, was referred by Dr. William Palmer Lucas on account of a persistent pyuria which had existed for the last three and a half years, ever since an attack of pneumonia. During this time he had repeated attacks of fever of a few days' duration, usually from two to three weeks apart. Recently, he had enuresis and marked frequency of urination. The urine was loaded with pus and showed motile rods and a faint trace of albumin; the phenolsulphonphthalein output was 15 per cent for the first hour and 20 per cent for the second hour. Plain roentgenograms were negative. Cystoscopic examination showed an enlarged hypertrophied verumontanum with redundant mucous membrane above, somewhat plicated into folds, but no definite valve formation. The bladder wall was not trabeculated, and there were no deep cellules. The right ureteropyelogram is shown in figure 13 and the left in figure 14. The verumontanum was partially destroyed by fulguration so as to remove the obstruction, and the ureters were catherterized and the kidney pelves lavaged on several occasions. The patient's general condition definitely improved. The total phenolsulphon-

phthalein output on Sept. 8, 1927, was: first hour, 55 per cent and second hour, 18 per cent. The urine showed from 40 to 60 pus cells, an occasional red blood cell and colon bacilli, but no casts. The following note was sent his father following the last examination:

"We have given Billy several conservative treatments in the hope that we would be able to relieve the progressive hydronephrosis that he has, but on checking up conditions today there seems to have been no improvement and I feel that something more radical should be done for him. He has a very advanced hydronephrosis of his right kidney and a markedly dilated and tortuous ureter on this side. The left side shows a similar condition but not nearly so marked. It would be possible

Fig. 13 (case 4).—Right ureterogram, which shows advanced degree of hydroureteral angularity and rather marked hydronephrosis, much more than on the opposite left side (fig. 14).

to relieve this obstructive condition by straightening out and shortening the ureter and if this is done at his time of life, one might get marked repair of the kidneys. If nothing is done, I think it certain that in time he will lose the right kidney because of the progressive hydronephrosis and that it would have to be removed and one would be fearful that the same progressive condition might continue on his good left side."

Operation.—On Oct. 6, 1927, the right ureter was exposed and found to be markedly tortuous and coiled on itself, the coils being plastered together with entering blood vessels and fibrous adhesions. Even after the ureter was completely

freed, it would immediately coil up, showing the points of marked secondary valvular-like obstruction because of the angularity. In order to relieve this, it was thought necessary to take out a section of the ureter, as was done in the previous case, and about 5 cm. of the upper third was removed and the two ends anastomosed. Attempts to follow the ureter down to the bladder through the lumbar incision encountered difficulty and after the anastomosis was completed, a catheter having been placed down the ureter through the kidney and pelvis by a nephrostomy opening, the patient was turned on his back and, through a right rectus incision, the lower ureter exposed extraperitoneally. A pronounced angularity was found here

Fig. 14 (case 4).—Ureteropyelogram of the left side, which shows a much less degree of back pressure change than the right side (fig. 13).

also. Retrograde catheterization met obstruction at the ureterovesical junction. Careful inspection revealed quite a marked valvelike condition at this point. About 7 cm. of this lower portion of the right ureter was resected and the end reanastomosed into the bladder, the ureteral orifice having been removed with the resected portion. This left a straight ureter. The right side was not disturbed.

The patient had quite a febrile reaction following the operation. The kidney was kept lavaged through the nephrostomy tube which was removed and reinserted several times before this opening healed. At the time of the lower ureteral operation, it was thought best to establish suprapubic drainage, and by means of the nephrostomy and suprapubic tubes lavage of the whole urinary tract on the left

side was possible. The nephrostomy tube was permanently removed on the twentieth day; following this there was quite a rise of temperature. A urethral catheter was inserted, and the temperature returned to normal a few days later. The nephrostomy opening quickly closed, and with the urethral catheter in place, suprapubic drainage had ceased by the twenty-sixth day. From now on for the next ten or twelve days, whenever the retention urethral catheter was removed, there was a rise of temperature which subsided with reinsertion of the catheter.

Fig. 15 (case 4).—Cystogram taken postoperatively, showing the straightened, though somewhat constricted, condition on the right side. The left ureter had been straightened at the time of operation but no resection done.

On the twelfth of November the boy's condition was markedly improved; he voided normally, though frequently; the total phenolsulphonphthalein output without the catheter for the first hour was 33 per cent and for the second hour 35 per cent. On November 26 a cystogram was taken (fig. 15) which showed that the left ureter was dilated in its upper third but that it was draining freely from the kidney pelvis. The patient was discharged from the hospital on December 8,

sixty-three days following his operation, and he had been completely afebrile for the last ten days before discharge. His operative wounds had all healed completely, and he was voiding normally. He had a normal renal function, but the urine still showed numerous pus cells. His general condition at the time of discharge was good.

He returned to the office for observation in May, 1928. In appearance he was a different boy than before operation; he had gained weight, and his general condition was improved. The total amount of phthalein in two hours as shown by the test was more than 60 per cent.

CASE 5.[5]—*History.*—K. D., a boy, aged 2 years, was referred by Dr. Langley Porter for examination on account of continual dribbling of urine and pyuria. He had been born at full term, delivery being normal. The feeding and developmental histories were normal. He had measles at the age of 2 months. Otherwise, the family history was unimportant. Since birth the mother had noticed that the patient could not void with a normal stream of urine but that there seemed to be a continual dribble. At the age of 6 months, a slight bloody urethral discharge was noted for the first time. This occurred again at 9 months. The child had always been fussy and irritable, crying while attempting to void. Cystoscopy and pyelography revealed a valvelike obstruction in the posterior urethra with hydro-ureters and hydronephrosis.

Physical Examination.—The child was anemic, sallow and undernourished; he was irritable and fussy, which made examination difficult. Marked puffiness of the face was present. The head was otherwise normal. The heart, lungs and abdomen were normal. There was no evidence of edema of the extremities. Examination of the blood showed: hemoglobin, 55 per cent; red blood cells, 3,300,000; white blood cells, 16,200; polymorphonuclear, 71 per cent; small leukocytes, 21 per cent; large mononuclears and transitionals, 8 per cent. On examination, the urine was pale yellow and normal, with a specific gravity of 1.002; it did not contain sugar and only a faint trace of albumin. Microscopic examination revealed numerous white blood cells and motile rods, an occasional red blood cell, but no casts. The phthalein output was: first hour, 7 per cent; second hour, 20 per cent; total, 27 per cent.

With a catheter in each ureter a right pyelogram was made which showed a markedly dilated and tortuous ureter and atrophic kidney on the right side. The left catheter was coiled up several times in the lower left ureter, indicating a large ureter on that side.

Operation.—Suprapubic cystotomy was performed with the patient under gas and oxygen anesthesia. The valves attached to the verumontanum and the posterior urethra were split. A bar formation at the vesical neck was excised. The left ureteral orifice was found gaping. The right ureteral orifice was slit open and several ounces of heavy purulent urine allowed to escape. Drainage was instituted through a tube drain and the wound closed in the usual manner.

It was possible to follow this boy closely from the time when the obstruction was removed from the lower tract, and the renal function increased from 27 to 65 per cent (February, 1924). There was a gradual loss of function so that within the last few months (1928) only a faint trace of phenolsulphonphthalein would be found on repeated tests in two hours, but on Jan. 9, 1928, the blood urea showed a nonprotein nitrogen of 42.2 mg. per hundred cubic centimeters. On reexamination at this time the right pyelogram showed a markedly dilated and tortuous ureter

5. Case 5 is the same as case 3324 in Hinman, Frank (footnote 1, p. 82).

and a pelvis and kidney shadow suggestive of secondary renal atrophy (fig. 16). The catheter in the left side was curled up in a small area (fig. 17). On account of there not being any nitrogen retention, it was thought that there might be some chance of repairing the left kidney by relieving the ureteral obstruction due secondarily to loops and angularities, as has been done in previous cases. The right ureteropyelogram indicated a secondary renal atrophy much like that found on the right side in another case, in which nephrectomy was performed. The parents were advised that operation might delay or improve an otherwise fatal malady.

Fig. 16 (case 5).—Right pyelogram showing a markedly dilated and tortuous ureter and a pelvis and kidney shadow suggestive of secondary renal atrophy.

Second Operation.—In January, 1928, the peritoneum was dissected back, exposing the kidney and ureter, the latter being markedly dilated, tortuous and angular. The coiled condition in the upper third was relieved by carefully dissecting off the adhesions which bound it, and then the ureter was carefully dissected free through this lumbar incision to its entrance into the bladder. There was a similar markedly tortuous and coiled condition in the lower third of the ureter, densely bound with adhesions which had to be cut before the ureter could be straightened. After the

ureter was straightened at both ends, the most marked angularities seemed to be at the end portions; it was easily twice as long as a normal ureter. It was divided at the pelvis by an oblique incision, and the cut end could then be raised so that it reached without stretching above the left shoulder of the patient. The amount of ureter resected was 19 cm. (fig. 18) enough being left for anastomosis to the pelvis. At the time the ureter was divided for anastomosis, it was also cut obliquely so that the resuturing would be in an oblique direction and not circular. Before the ureter was anastomosed to its pelvis, a nephrostomy opening was made and a no. 12 catheter, well perforated at the end and about 12 cm. up, was passed into the pelvis and on into the ureter, so that the upper openings of the catheter

Fig. 17 (case 5).—Ureteropyelogram of the left side showing a markedly dilated and tortuous ureter with advanced hydronephrosis above.

drained directly from the pelvis; the catheter passing on into the ureter splinted the point of anastomosis. After the lower third of the ureter was straightened, catheters could be passed easily into the bladder.

Postoperative Course.—The following postoperative notes are given in some detail because this case merits most careful study. In this case a patient with a typical obstruction of the posterior uretral valve showed complete recovery of renal function after the removal of the valves, but two years later was on the verge of having uremia as a result of renal insufficiency.

First Day: The patient's temperature rose to 40 C. (104 F.) rectally. The general condition was good; the fluid intake was 3,400 cc. and the measured output,

1,400 cc. The patient took fluid well by mouth. The nephrostomy tube drained well and was irrigated twice that day with 1 : 10,000,000 potassium permanganate solution.

Second Day: The highest rectal temperance was 39 C. (102.2 F.). There was slight serohemorrhagic drainage through the extrarenal rubber drain. The nephrostomy tube was draining well, and the patient's condition was satisfactory.

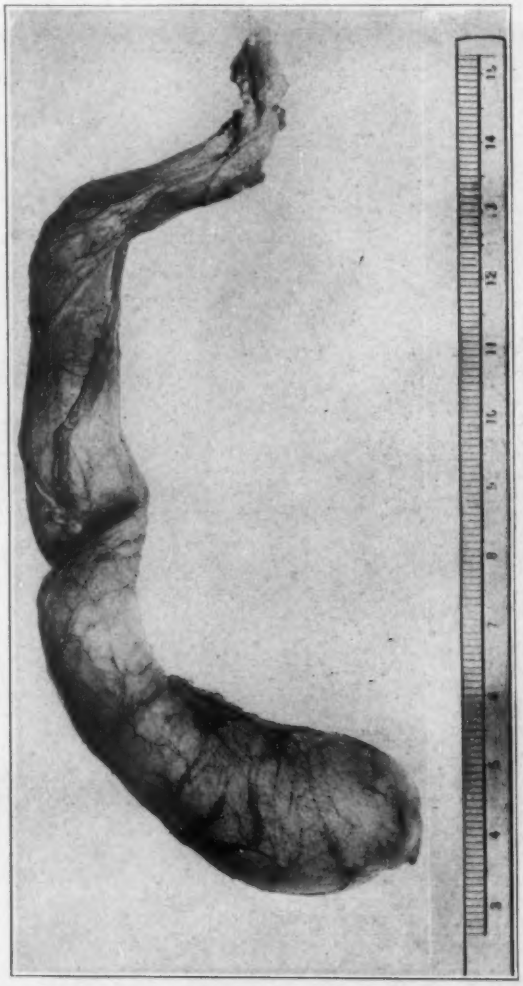

Fig. 18 (case 5).—Portion of ureter resected from the upper portion. The pronounced dilatation and elongation are at once obvious. This amount resected is about the same length as that left in, as shown in figure 19.

Fourth Day: The highest temperature was 38.6 C. rectally (101.4 F.); the pulse rate was 110; it was regular and of good volume. The patient occasionally voided small amounts of urine by way of the urethra, and still a slight amount of drainage occurred at the site of the extrarenal drain tube. The nephrostomy tube

drained well. The extrarenal tube was pulled out about 4 inches (10.1 cm.) and cut off flush with the wound. After intralumbar injection of phthalein the urine collected through the nephrostomy tube showed: first hour, 140 cc., 24 per cent; second hour, 130 cc., 15 per cent; total, 270 cc., 39 per cent. This is a most remarkable return of function four days after free drainage of the kidney had been established; all previous tests for many months before operation had shown only a trace or no phthalein at all.

Fig. 19 (case 5).—Ureteropyelogram of the left side taken postoperatively. The point of anastomosis of the upper part of the ureter that was sutured to the pelvis in an oblique manner is seen to be quite patent. However, there seems to be some constriction of the lower portion of the ureter as it enters the bladder and in this case it will be necessary to perform dilatations of this portion in order to get freer drainage. The author now believes that it would have been preferable to have resected the necessary amount of this elongated ureter at its lower portion so as to have thoroughly removed this constriction at its vesical end. Compare with figure 17.

Fifth Day: The drainage tube outside the kidney was removed; the nephrostomy tube was draining well; it had been irrigated with protargin mild

solution to determine whether the irrigation fluid passed on to the bladder. Following irrigation with this solution through the nephrostomy tube the patient urinated, and the urine voided was highly stained with the protargin mild solution, showing free drainage through to the bladder.

Eighth Day: The patient's temperature had been about 37.8 C. (100 F.) for the last four days; his general condition was remarkably good; he ate well, took fluids freely, seemed comfortable and contented and his appearance was altogether different from that previous to operation. The nephrostomy tube was draining well.

Ninth Day: Urine collected through the nephrostomy tube showed a phthalein return of: first hour, 21 per cent; second hour, 15 per cent and total, 36 per cent.

Fourteenth Day: The wound had healed except at the nephrostomy opening where the tube was still attached.

Sixteenth Day: Cystoscopy was performed, and ureteral catheters were placed on each side. These catheters were retained and were irrigated with protargin mild solution every two hours.

Nineteenth Day: The ureteral catheters were removed and a urethral retention catheter placed. The phthalein test showed: first hour, a return of 25 per cent; second hour, 20 per cent and a total of 45 per cent.

Twenty-First Day: The urethral catheter was removed.

Twenty-Third to Ninety-Second Day: Retention ureteral catheters were placed; they were irrigated every two hours as before, this time with mercurochrome-220 soluble. Connell suction was applied to the ureteral catheters, and they were removed on the twenty-seventh day. A retention urethral catheter was placed on the twenty-ninth day and through and through irrigations instituted. Ureteral catheters were again placed on the thirty-ninth day. The urinary discharge from the nephrostomy tube had been purulent, and the temperature during the last few days had been between 39 and 40 C. (102.2 and 104 F.). The total phthalein output on the forty-second day was somewhat diminished, there being a total of only 21 per cent. On the fifty-eighth day the hemoglobin was 45 per cent, the red blood cells 2,864,000, and the white blood cells, 7,860; the systolic blood pressure was 94; diastolic, 56. The patient was given a transfusion of 195 cc. of citrated blood without reaction; again on the sixty-sixth day he was given another transfusion of 100 cc. of citrated blood without reaction. The condition was now satisfactory. He had not had a rise in temperature for nine days, and he took fluids well and ate well. On the sixty-eighth day, the hemoglobin was 65 per cent; the red blood cells numbered 3,472,000 and the white blood cells, 7,900. The nephrostomy tube was removed on the sixty-fourth day, and all drainage tubes and catheters removed. The patient was discharged on the ninety-second day after operation. The temperature had been normal since the sixty-fifth day except for an occasional slight rise above 37 C. (98.6 F.).

The patient was seen several times after he returned home; his general condition remained satisfactory, and he did not have any febrile attacks. The urine was clear but still showed some pus. The last phthalein test on June 15, 1928, showed: first hour, 100 cc., 20 per cent; second hour, 100 cc., 6 per cent, a total of 26 per cent. Figure 19 shows the postoperative condition by ureteropyelogram.

CASE 6.—*History.*—F. T., a boy, aged 7, was seen in June, 1927, by Dr. Langley Porter and admitted to the pediatric service of the University Hospital for observation. His main complaints were loss of weight, exhaustion, hay-fever and asthma. Cystoscopy was performed in January, 1927, by Dr. Meads of Oakland on account of a persistent pyuria, and the patient wore a retention urethral catheter from September until May. Since birth he had had periodic attacks, lasting from two to three days, of loss of appetite with vomiting and a rise of temperature to 104 F.

or over. A cystogram was taken in June, 1927 (fig. 20), demonstrating the bilateral hydro-uretal angularity with hydronephrosis. A phthalein test in July showed a return of 6 per cent the first hour and 10 per cent the second hour, a total of 16 per cent. The urine was cloudy, loaded with pus and some red blood cells. Examination of the blood showed: 80 per cent hemoglobin; red blood cells, 3,930,000, and white blood cells, 12,400. The phthalein test on July 13, 1927, showed a return of 7 per cent the first hour and 19 per cent the second hour, a total of 26 per cent. During this stay in the hospital the patient had several marked

Fig. 20 (case 6).—Cystogram showing bilateral hydro-ureteral angularity with hydronephrosis.

febrile attacks, lasting three or four days, the temperature rising to 40 C. (104 F.). Following his discharge from the pediatric service, the patient was instructed to return weekly for ureteral catheterization and pelvic lavage. This was kept up rather regularly, but there was no improvement in the general condition, only slight improvement in function. The parents were advised that the ureters should be straightened surgically.

The patient was readmitted to the hospital on the urologic service on May 30, 1928. The phthalein output showed decided improvement following ureteral catheterization and pelvic lavage, showing on the day of admission a return of 30 per cent the first hour and 21 per cent the second hour, a total of 51 per cent.

Operation.—On May 31, 1928, with the patient under gas and oxygen anesthesia, a left nephrostomy and bilateral lower ureteral resection with vesical implantation were performed. He was placed on his right side as for an operation on the kidney, and the lower pole of the kidney was exposed and the ureter freed of its folds and adhesions in its upper portion. Through a small incision in the pelvis, the nephrostomy probe was pushed back through the thinnest portion of the kidney at its middle so that the nephrostomy tube would drain directly through the lumbar incision. A catheter tip that had been perforated was tied to the end of the probe and drawn back through the kidney pelvis and then threaded on down the ureter so as to splint its upper portion. After the lumbar wound was closed, the patient was placed on his back and a long midline incision made, the bladder being exposed. By careful stripping of the peritoneum, the ureter was successfully exposed and straightened on each side all the way to the kidney pelvis. About 8 cm. of the lower portions of each ureter was removed and the remaining ends implanted into the open orifices left after the ureterovesical end was resected. Catheters were threaded inside the bladder up these dilated ureters for purposes of drainage and a pessar catheter left in suprapubically through the cystotomy opening. The bladder was closed about these tubes.

The postoperative condition was satisfactory, although during the first twenty-four hours the drainage through the tubes was not satisfactory. After the second day the tubes drained well, and the patient's general condition at this time was satisfactory. He returned for observation three months after discharge, in excellent general condition, with a clear urine and a total phthalein output of more than 60 per cent in two hours.

OPERATIVE PROCEDURES IN CASES 1 AND 2

CASE 1.—The patient (previously described in this paper as case 1) was readmitted for operation on the lower ureters on May 29, 1928. The phthalein test on admission was: first hour, trace; second hour, 6 per cent. On May 30, the test showed: first hour, trace; second hour, 5 per cent. On June 1, the output was: first hour, 5 per cent; second hour, 3 per cent, a total of 8 per cent. The urine was fairly clear, with a specific gravity of 1.010; it showed a faint trace of albumin, no acetone or diacetic acid, and there were from 4 to 5 pus cells in each high dry field, but no red cells.

Operation was performed on June 2, 1928, under gas and oxygen anesthesia. On account of previous plastic operations at the ureteropelvic juncture, it was not considered necessary to perform nephrostomy. Through a midline suprapubic incision, the peritoneum was stripped back on each side and the markedly dilated hydro-ureters exposed and freed of all adhesions and angularities. It was necessary to ligate and divide the deep epigastric vessels on each side in order to make this exposure satisfactorily. The round ligament and the uterine vessels were exposed extraperitoneally and the ureter freed beneath and pulled up below these structures. There was definite narrowing of the ureter in its vesical portion so that one could pass a probe from above into the bladder with difficulty. The ureterovesical ends were resected, and the ureter, markedly elongated, after having been straightened was drawn through the opening in the bladder. About 8 or 9 cm. of the lower end of each ureter was removed and implantation secured with no. 2 chromic catgut, a large, long, rubber drainage tube having been passed retrograde from the bladder up each hydro-ureter. These tubes were brought out suprapubically alongside a pessar catheter and the bladder sutured about these three tubes. Inspection of the bladder neck showed that there was quite a fold of mucous

membrane posteriorly in the nature of a median bar which seemed to be somewhat obstructive; this bar was removed with the radio knife just as is done in the suprapubic operations for median bar, the bleeding points being tied off or thrombosed with the coagulation current. The patient's condition following operation was satisfactory.

CASE 2.—The patient (previously described in this paper as case 2) was readmitted for operation on the lower ureter of the remaining kidney because of continued febrile attacks, gradual loss of function and the fact that the ureteral hypertrophy and angularity and hydronephrosis were definitely progressive, as demonstrated by the recent ureterogram shown in figure 10. The phthalein output on the day of admission was: first hour, 50 cc., 5 per cent; second hour, 70 cc., 9 per cent, a total of 14 per cent.

Operation was performed on June 2, 1928, under gas and oxygen anesthesia. A midline suprapubic incision was made as in the previous case, and the peritoneum on the right side was stripped back after division of the deep epigastric vessels, a markedly dilated and angular ureter being exposed. This ureter was freed of its adhesions, straightened and drawn out below the round ligament and uterine vessels, as already described. By careful dissection its entrance into the bladder could be demonstrated and was found to be quite markedly constricted, so much so that even after a small incision was made in the dilated ureter just above the bladder it was not possible to pass a no. 6 or 7 probe into the bladder. The ureterovesical end was resected, a round opening being left at this side of the bladder, through which the elongated ureter was drawn, it being straightened above. It was implanted with chromic no. 2 sutures without tension, which left about 7 or 8 cm. projecting in the bladder. This was removed, a slit opening of that portion of the ureter projecting intravesically being left. A long no. 14 rubber drainage tube was threaded back through the bladder up the dilated ureter to its pelvis and brought out alongside of a pessar catheter in the bladder as in the previous case, the bladder being closed tightly about these two tubes. The bladder neck in this child showed a barlike condition, similar to that in case 1, which was resected with the radio knife as in the previous case.

The surgical treatment given these three boys and three girls has many points for contemplation and study. Will the right side in case 4, which has been untreated, improve or deteriorate? There is now no obstruction of the lower tract and it should improve, but hydronephrotic atrophy was progressive in the short period before the left side was operated on as it was in the other two cases (5 and 6) after removal of the obstruction of the lower tract. Perhaps the valvelike condition found at the ureterovesical juncture on the left side in this case is a factor in the more advanced destruction on this side.

Will the remnant of ureter that has been isolated by resection of portions of its two ends survive and function? This radical measure seemed at the time the only solution in cases 3 and 4. But attention was focused too much on the conditions of the upper ureter. The possibilities of success would seem better when a portion of one end is removed, as in cases 5, 6, 1 and 2. One must look with suspicion on the uterovesical end, and, although resection of the ureteropelvic portion seemed necessary at the time in order to remove an obvious

obstruction, more recent experience leads to the belief that this is not necessary. The upper ureter can probably be satisfactorily straightened by merely freeing it of its adhesions if the slack is taken up by resection of the lower end.

Will the remarkable improvement that has been immediate in all cases be permanent? The initial removal of the obstruction of the lower tract benefited all similarly but only temporarily. The procedure in case 1 was unsuccessful though the immediate benefit was marked. In case 2, a nephrectomy was required subsequently to the initial operation. Have the surgical measures used in cases 3, 4, 5 and 6, and just recently in case 1 and on the remaining kidney in case 2, removed the cause of progressive hydronephrosis? The period since operation is too short in any of these to prove anything in this respect. One can say at least that none of these patients is in a more precarious condition than before operation, and in each one the improvement in renal function as indicated by the phenolsulphonphthalein test has been prompt and usually remarkable.

SURGICAL TREATMENT

In suggesting a surgical program of treatment in this group of cases, it is believed that nephrostomy and lower ureteral resection with reimplantation constitute the most logical and promising method. On account of the more advanced change on one side and severe infection, there will, of course, be occasional cases in which ureteronephrectomy is at once indicated and required. A number of children with a definitely unilateral obstructive condition, in whom nephrectomy seemed the logical procedure, have been seen. Abnormal vessels or bands at the ureteropelvic juncture or double pelvis and double ureter produce a simple hydronephrosis with or without secondary infection that clearly differs from the condition in the particular group under discussion. Indeed, the ureteral changes above an obstructing aberrant vessel in a recent case may be strikingly analogous to the more general changes under discussion. In cases showing obstructive hydro-ureteral angularity, whenever the retention catheter or surgical exposure of the kidney shows a fair amount of parenchyma, consideration of its preservation is in order, and the first and most valuable surgical procedure to effect this is a successfully functioning nephrostomy. It gives the free drainage during convalescence that may be essential to success, although it was omitted in reoperating in the last two cases, the reason being that in case 1 previous plastic operations had been performed at both ureteropelvic junctures and in case 2 nephrostomy had previously been performed on the remaining kidney. It was thought that these previous operations on the kidneys would complicate a quick and satisfactory nephrostomy. It seems well proved that the

pyeloplastic type of operation as first performed in case 1 does not meet the necessary requirements, and I consider it fortunate that it has been possible to follow this case for so many years and to know what the ultimate result is. It would also seem that resection of portions of the ureter at its two ends is unphysiologic. It is much better, provided satisfactory ureteral straightening can be effected, to resect and anastomose at one or the other end, preferable the vesical. Freeing the ureter of its adhesions and straightening it will not always relieve obstruction, as was found in cases 3 and 4, in which were definite obstructive conditions at both ends of the ureter due to angulations and adhesions. The ureter, of course, is known to get a rich blood supply at different levels, and the experience in the two cases in which portions of both ends were resected seems to show that the middle portion may receive sufficient blood even after isolation from its two ends. In freeing the kinks of their adhesions, however, one should be careful to preserve as much of the blood supply as possible, because at this time the complete procedure of treatment cannot have been definitely determined.

The postoperative stricture at the point of anastomosis of the upper ureter to the pelvis in case 3 indicates that the method of ureteral anastomosis requires careful consideration. Because of this experience the ureter was divided obliquely in one case so as to give a long line of suture at the point of anastomosis and to insure much less likelihood of stricture afterward. The fact that one is anastomosing a markedly dilated tube does not seem to be sufficient security against formation of stricture afterward. However, it has been possible to dilate the upper ureter in case 3, and I believe that the result in the end will be satisfactory.

From the experience so far at hand, the best surgical method of attack in the treatment for obstructive hydro-ureteral angularity may be outlined as follows:

Step 1.—With the patient lying on the side over a kidney pad, a short curved lumbar incision is made at the twelfth rib. By carefully stripping up the peritoneum, exposure of the lower pole of the kidney and its ureter is possible, and in most cases delivery of the lower pole is all that is required.

Step 2.—The kidney is first freed. A small opening is then made in the pelvis, through which the ureter and pelvis are explored by catheters, probes or the finger and the amount of potential renal parenchyma determined. It is not always necessary completely to deliver the kidney (previous functional tests are a better guide).

Step 3.—Nephrostomy.—With a curved forceps or a curved nephrostomy probe, which has been made for this particular purpose, an opening is made from the middle or upper major calyx to the surface of the kidney by gently forcing the blunt end of the instrument through the thinnest portion of the renal parenchyma, and the end of a fairly good sized rubber tube or catheter (12 to 14 F in diameter) seized in the clamp or tied on the end of the probe and pulled back into the kidney

pelvis and out the pyelotomy opening. If the child's condition is questionable, the operation can be stopped at this point, and one can wait to see the amount of improvement that nephrostomy drainage will secure before undertaking to remove the obstructing ureteral angularities. So far this has been necessary in only one of the cases, as this point had been determined by ureteral catheter preparation in all of the others, but ureteral catheterization was unsatisfactory or almost impossible in this one case (case 2).

Before deciding on the surgical steps to follow, one must first determine the obstructive conditions of the upper portions of the ureter. Previous ureteropyelograms or cystograms may have indicated what condition to expect, but one now has an opportunity to find out definitely. The whole ureter requires careful inspection and exploration. Because it is always possible that resections of portions of the ureter at both ends will be necessary in order properly to relieve obstruction, the blood supply to its middle third should be preserved, and this cannot be done safely if it is stripped free from above downward. But the peritoneum usually may be stripped off the anterior ureteral wall quite readily, all the way to the bladder, the ureter being left more or less undisturbed in its bed, and one can then free the tube in its upper and lower third and leave the middle portion alone until after inspection, if this seems advisable.

Sept. 4.—If it is decided that resection of a portion of the upper ureter will fulfil the requirements, then the middle third must be freed, irrespective of its blood supply, because it is only by traction and straightening that the obstructive angularities of the other end are removed. This would be impossible with the middle third fixed in position.

The ureter is divided obliquely at or near the pelvis and the length to be resected determined by pulling the unattached end up over the kidney. This redundant portion is then removed, also by an oblique incision, so as to give a long line of suturing at the site of anastomosis. The nephrostomy catheter previously placed is perforated at two points, the first near its end, the second at a good distance up (from 10 to 15 cm.); the catheter is then passed on down the ureter so that the higher perforations come to lie in the kidney pelvis, and the long end of the catheter which passes down the ureter splints it at the site of anastomosis. In those cases in which there is definite abnormality at the ureterovesical end that cannot be corrected through the lumbar incision or that requires resection, I believe it advisable to wait until the child has fully recovered from this first operation. As long as the nephrostomy tube drains well, the obstructions of the lower ureter are of no clinical importance but must be remedied, of course, before one can expect benefit to result. The nephrostomy opening may fail to close otherwise. This next step or operation is best performed through a midline suprapubic incision, as described in step 5.

If, after ureteral inspection and exploration, one decides that resection of a portion of the lower third will effect straightening of the middle and upper angularities and thus relieve their valvelike obstruction, and I believe that this procedure is much to be preferred, then after straightening the upper ureter and after the nephrostomy tube has been adjusted in the pelvis and the ureter, as already described, the lumbar wound is closed. It will usually be advisable to perform the next step as a second stage later.

Step 5.—The patient is placed in slight Trendelenberg position, and the bladder is exposed and the peritoneum dissected back at the side so as freely to expose the lower ureter. The ureter is freed, opened and thoroughly explored. I am convinced of the importance of this step because defects at the ureterovesical end must be common even though they have been demonstrated definitely in only three patients: one with a ureterovesical cyst, one with a ureteral pouch and another with constriction. Because of the marked elongation in these cases, simply freeing the ureter of its adhesions and straightening it will fail unless the slack is taken up by resection at one or the other portion. The ureter would simply coil up and become adherent again. Because of the significance of conditions at the vesical end as well as the more physiologic end-result, resections here should be given preference, and in most cases complete resection of the ureterovesical orifice is indicated.

SUMMARY

The surgical method of attack in the treatment for obstructive hydro-ureteral angularity may be summarized as follows:

First Stage.—Nephrostomy is performed on both sides or on the more diseased side, the upper ureters straightened and freed and then splinted with the nephrostomy tubes.

Second Stage.—The bladder and lower ureters are exposed through a midline suprapubic incision. Each ureter is straightened and freed either to its pelvis or, when nephrostomy has been done, to the free portion above. Enough of the lower portions are resected to take up the slack and, after the bladder is opened, they are implanted in the opening into the bladder made by resection of the old ureterovesical orifice. Large catheters are placed well up the ureters as splints and for drainage and lavage. The vesical neck is thoroughly inspected and any bar or contracture that is present is resected. The bladder is closed about a suprapubic drainage tube and the ureteral catheters.

ABSTRACT OF DISCUSSION

DR. ROBERT H. HERBST, Chicago: I was particularly interested in Dr. Hinman's report, especially that part which referred to the failure to obtain a good result by ureteropyeloplasty in the relief of hydronephrosis, due to obstruction at the ureteropelvic junction. My associates and I performed this operation on thirty dogs to determine why this procedure so frequently fails to relieve the hydronephrosis. The technic was as follows: A short time after hydronephrosis was produced by narrowing the ureteropelvic junction, a technic similar to the Heineke-Mikulicz procedure used for the relief of pyloric stenosis was carried out on the ureteropelvic junction. A longitudinal incision was made which was sutured transversely. The dogs were killed later and the kidney studied. We found that the operation failed to relieve the hydronephrosis in nearly all these dogs, and this was due to a folding over or buckling of the part of the ureteral wall opposite the line of suture. This prevented normal drainage from the renal pelvis. Christian Fenger was the first to describe this operation (1898), and he and some who followed him reported good results, based on the fact that the patients were relieved of pain. However, in later years, following the advent of pyelographic and functional study, the results from this procedure have been found far from satisfactory. We believe

that it is better to cut the ureter off at its junction with the pelvis and reimplant it higher up into the wall of the pelvis. This not only does away with the obstruction at this point but has a tendency to straighten out the ureter and to take care of much of its angulation.

Dr. Budd C. Corbus, Chicago: In looking over the literature one is struck by the scarcity of reports of such work as that of Dr. Hinman in children. I wish to ask those who do this work to record their observations on the cystoscopic examinations. Much is heard about the instrumentation of children. I should like to hear Dr. Hinman tell about his methods. I have been in the habit of using pantopium hydrochloricum in this work. This can be given to children up to about 4 years of age in doses of ⅙ grain (0.007 Gm.) hypodermically and to children from 6 to 9 years of age in doses of ⅛ grain (0.01 Gm.); after children reach the age of 9 years, ⅓ grain (0.02 Gm.) may be given. Judging from the necropsy reports published, I am of the opinion that the children with infection of the upper urinary tract die early in life. I believe that most of the children with chronic urinary infection from 4 to 6 years of age have obstructions of either the vesical neck or the posterior urethera. A cystogram and a bougie, together with a determination of residual urine, will often tell where the obstruction is. Everyone is not as expert a surgeon as Dr. Hinman, and plastic work on the ureter in children is difficult. If an indwelling catheter was put into the bladder of these children, I wonder whether one could not achieve in time some of the things he accomplishes surgically.

Dr. William F. Braasch, Rochester, Minn.: These cases of ureterectasis present a difficult problem, and the improvement Dr. Hinman has demonstrated is a hopeful step. There are two factors in the etiology of the dilated ureter, namely, obstruction and atony. I have long been interested in the atonic type, which I formerly called inflammatory dilatation and regarded as being the result of cicatricial changes in the ureteral wall. Atony of the ureter with dilatation may result from interference with peristalsis. This has been shown to be possible by Beach, who demonstrated in animals that there may be as much dilatation below a cannula inserted in the ureter as above it. I am particularly interested in those cases of ureterectasis Dr. Hinman referred to as occurring in girls in whom there was no obstruction at the ureteral orifice or at the bladder neck. It is possible that both atony and cicatricial obstruction may be present in localized areas of the same ureter and may act in a vicious circle on each other. In the group in which atony predominates, it would hardly seem possible that any operative procedure would be of much value. This has been observed by Russian clinicians, who divide cases of hydronephrosis into two classes, those due to obstruction and those due to atony, and they advise operation for the former condition but not for the latter. It is evident from Dr. Hinman's experience that in cases of ureterectasis in children a plastic operation may remove the obstruction which is secondary to angulation of the ureter, and even though peristalsis could not be completely restored the drainage from the kidney was greatly improved. This subject is of great interest. I feel that the atonic element has been overlooked in the past, and that in the future it will be a much more important factor in the treatment of the obstructed ureter.

Dr. William E. Stevens, San Francisco: Pediatricians are beginning to realize that the urologist can often render valuable assistance in the diagnosis and treatment of these patients. It is a simple matter to obtain a catheterized specimen of bladder urine, even in infants, by means of an olive tip or Blasucci ureteral catheter. The presence of pus or blood always demands investigation of the kidneys and ureters. Cystoscopy and ureteral catheterization are not difficult in

infants or children, although in young children the bladder and, consequently, the ureteral orifices are at a higher level. In females the bladder also lies deeper in the pelvis. As a rule, there is little reaction following these procedures. Operative measures are also tolerated well during infancy and childhood if care is taken to prevent excessive loss of blood. In 232 pathologic conditions of the urinary tract seen in the pediatric service of the Stanford University Medical School, renal calculi were present in only three instances. They were bilateral in one of these cases. Ureteral calculi are rare, but calculi in the bladder and the urethra are more frequently reported in the literature. Fifty per cent of the uncomplicated cases of pyelitis cleared up under conservative treatment. Almost 100 per cent became microscopically negative and 80 per cent culturally negative following one or more ureteral catheterizations with drainage and irrigations of the renal pelvis. I should like to emphasize the fact that modern urologic methods of diagnosis are always indicated in these children.

Dr. George R. Livermore, Memphis, Tenn.: Dr. Stevens said that 20 per cent of his cases cleared up under medical treatment. What is meant by "clearing up?" In many cases nothing is found in the urine to call attention to pathologic changes in the kidney. I recall the case of a child, about 7 years of age, in whom the urinary tract had never been considered, because there were no urinary symptoms. This child had recently had measles. There was no albumin in the urine, and the family physician thought that the patient was all right. The only reason the child was brought to me was because he fell and struck the suprapubic region on a rock. This caused blood to appear in the urine. On cystoscopic examination I found greatly dilated ureteral orifices, as large as my finger or larger. I could not get the catheter up far before I encountered obstruction. There was great tortuosity of the ureters and the catheter would not pass, but by filling the child's bladder with a pyelographic medium and tilting the table in the Trendelenberg position, I found what was wrong. In some cases in which marked pathologic changes are present in the kidneys urinary symptoms are absent; hence, one must be careful in saying "the condition has cleared up."

Dr. Albert E. Goldstein, Baltimore: My impression was that when these obstructions were dilated relief was always obtained, irrespective of the type of obstruction. I was under the impression that when a great deal of destruction of the kidney is present, irrespective of what is done, the function of the kidney is not improved but drainage with less chance for infection is allowed. Dr. Hinman cited several cases in which prior to operation the function was 4, 5 or 6 per cent, and in which within from forty-eight to seventy-two hours after operation there was increased function of from 7 to 28 per cent. It hardly seems possible to improve the function so rapidly. It is my opinion that when the first functional test was made the obstruction was probably transvesical and that when the second test was made the catheter was in the kidney, permitting the outflow of urine which prior to that time had remained in the kidney, due to stasis. Likewise, I was under the impression that Dr. Hinman had written several years ago that when one finds hydronephrosis produced by obstruction rarely does the patient recover.

Dr. Joseph S. Eisenstaedt, Chicago: Several years ago I reported a case similar to that of Dr. Livermore, describing it as a congenital megalo-ureter, without primary obstruction. I have had three cases in little girls. One case came to autopsy, the other two children are living; there was no sign of obstruction in the one who died. The point of importance is that there is no obstruction in the urinary tract. The surmise as to their cause is interesting. Dr. Braasch spoke of the atonicity, and that describes it as well as any other term. In my article at that

time, I said that it might be an embryologic type of ureter. The musculature of the ureter starts to develop at about the sixth week of intra-uterine life. It is obvious that with this type of case a surgical procedure will be of no avail. The lack of muscle tone in such a ureter may be due to a peculiar congenital neuropathy. There is at least a likelihood of an abnormal nervous mechanism in these cases.

Dr. Clarence G. Bandler, New York: I should like to cite the case of a girl, aged 10, the daughter of a physician, who came under my care last year. This child had been ill continuously for the past four years, and at intervals prior to that time. She had been treated during the entire period by a gastro-enterologist for attacks of so-called "cyclic vomiting." At no time, apparently, did she have subjective symptoms referable to the urinary tract. Finally, after four years of observation, the physician had a roentgenologic examination of the gastro-intestinal tract conducted, and in the course of this study a large shadow was noted in the left renal area. The child was then referred to me, and my examination revealed a cluster of calculi in the left renal area, although a kidney shadow was not discernible and there was no evidence of left renal function. The right kidney was functioning in a normal manner. I operated on the child and removed what constituted her left kidney, which was only a thin fibrous capsule enclosing a number of small, regularly faceted calculi and which gave one the impression of an aplastic kidney replaced by multiple calculi.

Dr. Francis H. Redewill, San Francisco: I am interested in etiology from the standpoint of the prevention rather than the attempted treatment for the existing lesions. A number of years ago, Osborne and Mendel showed that if vitamin A is withdrawn from the diet of guinea-pigs the animals develop calculi and ulcers in the bladder within a short time. McCarrison showed that the natives of Africa did not have calculi or ulcers if they were on a natural diet consisting of milk, nuts and fruit. The same was true of monkeys, but if these foods were taken away and the natives and animals were given polished rice almost exclusively they quickly developed calculi and ulcers. It is possible that due to lack of calcium these persons may develop cicatricial obstruction and also calculi. There are cases on record in which the administration of parathyroid extract has cured buccal and duodenal ulcers. My associates and I conceived the idea of administering parathyroid extract and the vitamins. One of the physicians working in San Francisco has shown that a condition of symbiosis exists between the various vitamins and that one must use vitamins A, B, C, D and E in proper proportion if one wishes to prevent the formation of calculi in experimental animals.

Dr. A. I. Folsom, Dallas, Texas: For several years I have been doing 90 per cent of the cystoscopies on children under local anesthesia. It is not difficult. I wish to call attention to the silent pathologic changes in many of these cases. A child, aged 6, with a nephrostomy wound in the left side, on examination showed as large a hydronephrosis on the right side as on the left, but had no symptoms. I did a ureteropyeloplasty and obtained a satisfactory result. The child has remained well for nine years. I cannot understand how this boy has lived so long with hardly any kidney tissue. He is a pitcher on a baseball team, and can play for nine innings without difficulty. In regard to stones in children, the mothers often show stones, and the condition is familial. In those cases in which the stone can be demonstrated to be composed of cystin, in a large percentage of the cases the condition can be cleared up by alkalinization. I hesitate to "dip my oar" into the discussion Dr. Hinman has raised on the hypertrophied and angulated ureters. It has been a confusing subject for a long time. Dr. Braasch suggests the use

of the term atonic, but that does not solve the problem. If my understanding of atony is correct, these ureteral walls should show a diminution or thinning of the muscular coats, and yet, as shown in Dr. Hinman's illustration, the reverse is true; so I do not believe that one can wipe the slate clean by using the terms atony and destruction. Another thing that has not been touched on is the cause of this condition. One analogy I would like to suggest is that in all cases of extrophy of the bladder, in the cases of extravesical implantation of the ureter, the great majority show changes of the same type that Dr. Hinman reported. I wonder whether there is not an underlying stratum of disturbance of the sympathetic nervous system in these ureters.

Dr. Frank Hinman, San Francisco: If Dr. Goldstein will read the paper carefully he will see why one can expect to get a high percentage of renal repair in these cases.

ENDOMETRIOSIS *

KIYOSHI HOSOI, M.D.
AND
LOUISE H. MEEKER, M.D.
NEW YORK

That endometrium may be found at some distance from the uterus is generally accepted. The many explanations given have not been accepted as final, and for this reason additional observations are valuable. The seven cases reported in this paper add something to the present knowledge.

Our report includes cases of endometriosis (1) within and adjacent to an inguinal lymph node and in the transverse colon with malignant degeneration; (2) in the vermiform appendix; (3) in a laparotomy scar; (4) in the urinary bladder, and (5) of endometrial particles in the tubal lumen.

CASE REPORTS

CASE 1.—*Endometriosis in an Inguinal Lymph Gland Associated with Endometriosis of the Groin and Endometrial Carcinoma of the Transverse Colon.*

H. A. B., aged 30, unmarried, never pregnant, gave a normal menstrual history, menstruation beginning at 16 years. When 20 years old, the patient was operated on for a right inguinal hernia. The hernia returned in the scar which was swollen and tender, and especially painful before and during each menstrual period. The supposed hernia was again removed, six years after the first operation, and was found to be a cyst filled with bloody fluid. A pathologic report was not made. Relief was not obtained from this second operation and six months later the operative area was again swollen and painful, especially before and during menses with metrorrhagia. At this time a laparotomy was performed, and a "chocolate cyst" of the left ovary was found attached to the right broad ligament. In this broad ligament adjacent to the cyst, there was also a normal accessory suprarenal gland as shown by the microscopic section. A left salpingo-oöphorectomy and a shortening of both round ligaments were performed. It was doubtful whether the tarry cyst was of the endometrial type or not, as most of the lining was destroyed except a few tall cylindric cells and one or two secreting glands.

Nine years after the first operation, the patient was admitted to the New York Post-Graduate Hospital. She again complained of swelling and pain in the right groin, especially following each period. At operation, the inguinal tumor mass was found to be connected with the right round ligament. The entire inguinal growth was removed. It measured 42 by 32 by 21 mm., and in addition there was one inguinal lymph node, 12 by 6 by 6 mm. The larger specimen showed numerous cystic spaces filled with old and fresh clotted blood, embedded in a matrix of fibrous tissue (fig. 1). Here and there small brownish pigmented areas were

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* From the Department of the Laboratories, New York Post-Graduate Medical School and Hospital.

present. The microscopic examination showed numerous small and large cysts with a lining of endometrium, composed of branching tubules and stroma cells, similar to the miniature uterine cavity of Cullen (fig. 2). Many of these cavities were markedly distended with blood and contained portions of stroma with tubules (fig. 3). In sections stained by Perl's method iron pigment was demonstrated, especially in and around the endometrial islands. Smooth muscle fibers were also present around these glandular areas, as shown by the van Gieson stain.

Serial sections of the lymph node revealed five separate, nonbranching, straight gland tubules, ending blindly at both ends. Three of them were located beneath the capsule, the others farther inward (fig. 4). One subcapsular tubule was dilated into a small cyst and was lined by flat or low cuboidal cells, a number of which were ciliated. The lining cells of the other tubules ranged from tall columnar to low cuboidal with occasional cilia (fig. 5). The lumen was empty or contained granular material and exfoliated epithelial cells. Beneath the epithelium there was a thin layer of spindle cells which stained yellow by the van Gieson method. Underlying the spindle cells there were endothelial phagocytes, containing blood pigment granules, as proved by Perl's method.

Fig. 1 (case 1).—Cross-section of gross specimen of groin endometriosis showing the characteristic appearance of cysts and fibrous strands. The numerous various-sized cystic spaces are filled with old and fresh blood.

In 1928, the patient was readmitted to the New York Post-Graduate Hospital with symptoms of intestinal obstruction. Roentgenographic and fluoroscopic examinations showed complete obstruction in the midtransverse colon, at the level of the first lumbar vertebra. At operation, 20 cm. of the transverse colon was resected. An unusually severe hemorrhage followed. The obstruction was caused by a tumor mass, 4 by 4.5 cm., with nipple-like projections having a smooth surface (fig. 6). The lumen was constricted to a narrow slit. The entire thickness of the wall was involved in the neoplastic process. The only ulceration was at the base of the growth. Small firm nodular masses were present on the serous surface, to which tabs of omental fat were adherent. The omental fat contained eight metastatic nodules, with broken-down centers, the largest measuring 4 mm. in diameter. At operation, it was seen that the metastases in the liver and in the mesenteric nodes were hemorrhagic in appearance. A cut surface of the main tumor mass was somewhat soft and yellowish gray. In the involved wall of the colon there were numerous cystic spaces; the largest, 14 mm. in diameter, showed numerous papillary growths (fig. 7). The spaces contained coagulated or granular material which was occasionally deep brown.

Microscopic examination showed an adenocarcinoma forming the nipple-like projections mentioned in the description of the gross examination. These were covered everywhere by intestinal mucosa, indicating that the neoplasm was invading the mucosa from the deeper parts (fig. 8). In the ulcerated area where the tumor has replaced the intestinal mucosa, there was a sharp differentiation between the tumor cells and the epithelium of the mucosa. The dilated cystic spaces in the wall of the colon were lined by cells varying in form from very flat to very tall columnar. The latter were of irregular height so that the free borders of the cells were seen to form an irregular surface when viewed under higher magnification. In the large cysts were papillary masses of carcinoma. A number of the smaller

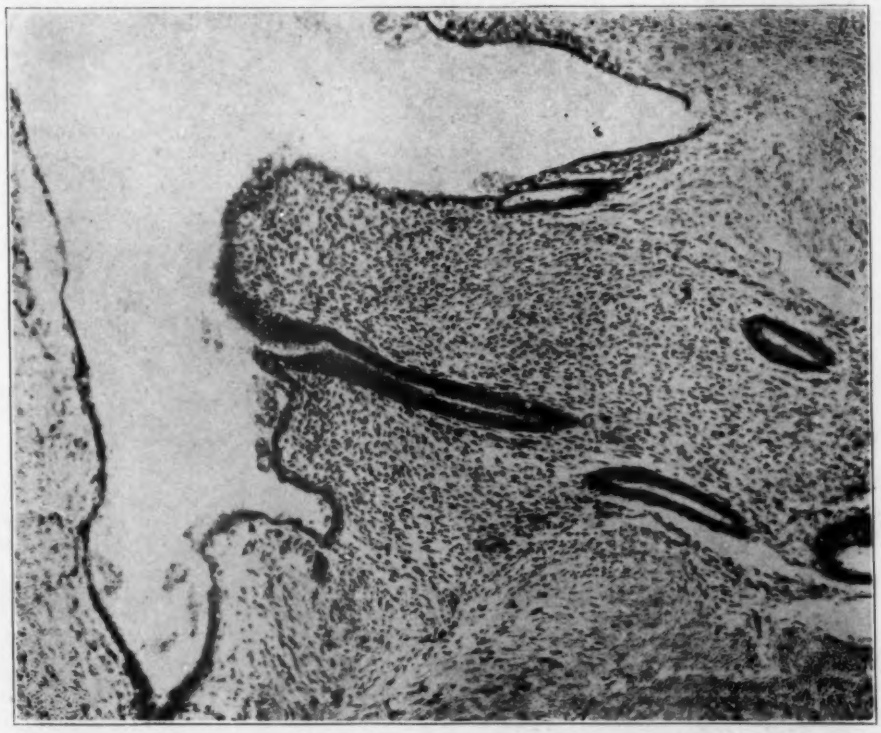

Fig. 2 (case 1).—Endometriosis of the groin. The elongated endometrial tubules are surrounded by definite cellular stroma, indistinguishable from that of the normal endometrium. The miniature endometrial cavity of Cullen is well shown. Medium power.

dilated cysts were surrounded by a more or less thick zone of lymphocytes. Persistent search was rewarded by the finding of a bunch of definite cilia on the flat epithelial lining in one of the dilated cysts (fig. 9).

Sampson [1] called attention to the fact that cilia are more easily found on the flattened epithelium of dilated gland lumina than on the tall columnar cells. We can confirm this by our observations.

1. Sampson, J. A.: Endometrial Carcinoma of the Ovary, Arising in Endometrial Tissue in That Organ, Arch. Surg. **10**:1 (Jan.) 1925.

Comment.—There are three accepted types of tumors of the round ligament: intraperitoneal, extraperitoneal and intracanalicular (in the canal of Nuck). We shall concern ourselves only with the extraperitoneal tumors, which have been interpreted variously as tumors of the groin, vulva or lower anterior abdominal wall. In 1896, Cullen [2] reported the first case of adenomyoma of the round ligament. In 1903,

Fig. 3 (case 1).—Endometriosis of the groin. This large distended cystic cavity is partially lined by ciliated cuboidal epithelium. Two large pieces of desquamated epithelium are present in the blood in the cavity. To the right and left of it are similar but smaller cystic spaces, containing blood. Low power.

2. Cullen, T. S.: Adenomyoma of the Round Ligament, Bull. Johns Hopkins Hosp. **7**:112, 1896; Further Remarks on Adenomyomata of the Round Ligament, ibid. **9**:142, 1898; Adenomyoma of the Round Ligament and Incarcerated Omentum in an Inguinal Hernia, Together Forming One Tumor, Surg. Gynec. Obst. **22**:258, 1916; The Distribution of Adenomyomata Containing Uterine Mucosa, New York State J. Med. **19**:295, 1919.

Emanuel [3] was able to collect from the literature seventy-six cases of round ligament growths, fifteen of which were myomas, fibromyomas or adenomyomas. He added four cases of his own, only one of which was an adenomyoma. Polster,[4] in 1926, found thirty-four extraperitoneal adenomyomas.

As regards pathogenesis, a number of theories have been advanced—Sampson's implantation theory,[5] Halban's theory of lymphatic dissemination,[6] von Recklinghausen's dysontogenetic theory of misplaced

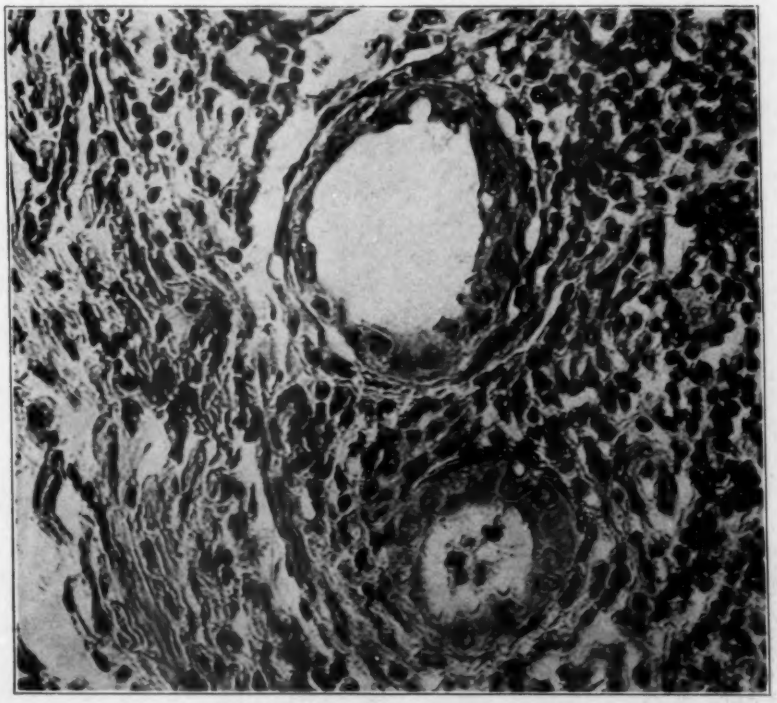

Fig. 4 (case 1).—Inguinal lymph node. Two epithelial tubules are present beneath the capsule which appears as irregular strands to the left. High power.

3. Emanuel, R.: Ueber Tumoren des Ligamentum rotundum Uteri, Ztschr. f. Geburtsh. u. Gynäk. **48**:383, 1903.

4. Polster, K. O.: Beitrage zur Kenntnis der heterotopen Wucherungen vom Bau der Uterrusschleimhaut, Virchows Arch. f. path. Anat. **259**:96, 1926.

5. Sampson, J. A.: Perforating Hemorrhagic (Chocolate) Cyst of the Ovary, Arch. Surg. **3**:245 (Sept.) 1921.

6. Halban, J.: Hysteroadenosis Metastatica: Die lymphogene Genese der sogenannten Adenofibromatosis heterotopica, Wien. klin. Wchnschr. **37**:1205, 1924; Berichte ueber geburtsh.-gynaek. Ges. in Wien., Nov., 1924; Zentralbl. f. Gynäk. **49**:387, 1925; Arch. f. Gynäk. **124**:457, 1925.

wolffian or muellerian duct,[7] and the serosal theory of Iwanoff[8] and Meyer.[9] Furthermore, Sampson[10] expressed the belief that metastases may occur by way of the veins, as shown by his injection experiments. Each of these theories offers an explanation as to the mode of origin of the different endometrial growths in case 1, which is peculiar on more counts than one, as it included epithelial tubules in an inguinal lymph node and adenocarcinoma of the endometrial type of the transverse colon, the latter an entirely new observation.

Fig. 5 (case 1).—Inguinal lymph node showing the ciliated cells lining the wall of another distended epithelial tubule. High magnification.

7. Von Recklinghausen, F.: Die Adenomyome und Cystadenome der Uterus- und Tuben-wandung, ihre Abkunft von Resten des Wolffschen Koerpers, Berlin, A. Hirschwald, 1896.

8. Iwanoff, N. S.: Druesiges cystenhaltiges Uterusfibromyom complicirt durch Sarcom und Carcinom (Adenofibromyoma cysticum sarcomatodes carcinomatosum), Monatschr. f. Geburtsh. u. Gynäk. **7**:259, 1898.

9. Meyer, R., in Veit: Handbuch der Gynaekologie, Wiesbaden, J. F. Bergmann, 1907, vol. 1, p. 413.

10. Sampson, J. A.: The Escape of Foreign Material from the Uterine Cavity into the Uterine Veins, Am. J. Obst. **78**:161, 1918; Metastatic or Embolic Endometriosis Due to the Menstrual Dissemination of Endometrial Tissue into the Venous Circulation, Am. J. Path. **3**:93, 1927.

Epithelial inclusions of the endometrial type in lymph nodes first described by Ries,[11] in 1897, have been observed rarely. Schauta[12] observed these inclusions in the preinguinal as well as in the inguinal nodes, and there are two other cases in inguinal nodes, those by Meyer[13] and by Lauche.[14] In the remaining reported cases, the sacral, the parametrial, the hypogastric, the iliac and the lumbar nodes were involved. Wertheim[15] found these inclusions in the regional lymph nodes in 13 per cent of his cases of uterine carcinoma; Schauta in 23.3 per cent. Von Franqué,[16] Manteufel[17] and others have also observed them in cases of uterine cancer. Ries, Wülfing,[18] Borst,[19] Sick,[20] Kroemer,[21] Wertheim, Luethy,[22] and Friedlander and Foot[23] thought that the inclusions were derivatives of the wolffian duct. According to Schauta, their presence in the preinguinal and inguinal nodes speak against the wolffian duct origin. On the other hand, Meyer, Kermauner and Laméris,[24] Falkner,[25] Brunet,[26] Sitzenfrey,[27] Gutfeld,[28]

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11. Ries, E.: Eine neue Operationsmethode des Uteruscarcinoms, Ztschr. f. Geburtsh. u. Gynäk. **37**:518, 1897.

12. Schauta, F.: Die Berechtigung der vaginalen Totalextirpation bei Gebaermutterkrebs, Monatschr. f. Geburtsh. u. Gynäk. **19**:475, 1904.

13. Meyer, R.: Epitheliale Hohlraeume in Lymphdruesen, Verhandl. der Ges. f. Geburtsh. u. Gynaek. zu Berlin, May 8, 1903; Ztschr. f. Geburtsh. u. Gynäk. **49**:554, 1903.

14. Lauche, A., cited by Halban, J.: Ber. der Geburtsh.-gynaek. Ges. in Wien, Dec., 1924; Zentralbl. f. Gynäk. **49**:664, 1925.

15. Wertheim, E.: Zur Frage der Radicaloperation beim Uteruskrebs, Arch. f. Gynäk. **61**:627, 1909.

16. Von Franqué, O.: Das beginnende Portiocancroid und die Ausbreitungswege des Gebaermutterkrebses, Ztschr. f. Geburtsh. u. Gynäk. **44**:173, 1901; Zur chirurgischen Behandlung des Uteruskrebses, Centralbl. f. Gynäk. **26**:1276, 1902.

17. Manteufel, P.: Untersuchungen ueber Metastasenbildung in den iliakalen Lymphdruesen bei Carcinoma uteri, Beitr. z. Beburtsh. u. Gynäk. **8**:253, 1904.

18. Wülfing, H.: Zur Pathologie der Geschwulstbildung im weiblichen Geschlechtsapparat, Ztschr. f. Geburtsh. u. Gynäk. **44**:1, 1901.

19. Borst, M.: Die Lehre von den Geschwuelsten, Wiesbaden, J. F. Bergmann, 1902, p. 216.

20. Sick, K.: Flimmerepithelcysten in der Nebennierenkapsal und in einer Beckenlymphdruese, Virchows Arch. f. path. Anat. **172**:468, 1903.

21. Kroemer, P.: Klinische und anatomische Untersuchungen ueber den Gebaermutterkrebs, Arch. f. Gynäk. **65**:626, 1902.

22. Luethy, F.: Ueber angeborne Epitheleinschluesse in Lymphknoten, Virchows Arch. f. path. Anat. **250**:30, 1924.

23. Friedlander, A., and Foot, N. C.: Report on a Case of Malignant Small-Celled Thymoma with Acute Lymphoid Leukemia, Am. J. M. Sc. **169**:161, 1925.

24. Kermauner, F., and Laméris, H.: Zur Frage der erweiterten Radikaloperation des Gebaermutterkrebses, Beitr. z. Geburtsh. u. Gynäk. **5**:87, 1901.

25. Falkner, A.: Zur Frage der epithelialen Hohlraeume in Lymphdruesen, Centralbl. f. Gynäk. **27**:1496, 1903.

26. Brunet, G.: Ueber epitheliale Schlaeuche und Cysten in Lymphdruesen, Ztschr. f. Geburtsh. u. Gynäk. **56**:88, 1905.

and Schiller [29] expressed the belief that they arise from the lymphatic endothelium through irritation from chronic inflammation or from the toxic action of the associated uterine carcinoma. Brunet and Sitzenfrey both described a "transition" of lymph channels into epithelial tubules.

Fig. 6 (case 1).—Gross specimen of endometrial carcinoma of the transverse colon. The mammillated appearance, produced by the carcinoma, pushing the mucosa forward, is clearly shown.

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27. Sitzenfrey, A.: Ueber epitheleliale Bildungen der Lymphgefaesse und Lymphraeume in Beckenlymphknoten bei Uteruskarzinom und bei karzinomfreien, entzuendlichen adnexerkrankungen, Ztschr. f. Geburtsh. u. Gynäk. **57**:419, 1906.

28. Gutfeld, V.: Inaug. Diss., Berlin, 1913; cited by F. Luethy (footnote 22).

29. Schiller, W.: Zur Frage des ektopischen Endometriums, Arch. f. Gynäk. **127**:544, 1925-1926.

Schindler [30] and Halban attributed their presence to dissemination through the lymphatics from the uterus, and Sampson added the venous route. All these authors, except Lauche, failed to mention the presence of blood pigment which we demonstrated in case 1. In this case, the narrow spindle-cell zone around the inclusions stains yellow, like smooth muscle, by the van Gieson stain. Stroma identical with that seen in the normal endometrium in the uterus has not been observed in lymph nodes, except by Lauche, who mentioned definitely that he found here and there in an inguinal node a suggestive cellular stroma. Sampson [31] described the invasion of the lymphatic channels by endometrial polyps and suggested that they may metastasize through these channels to the groin. Ewing [32] stated that the inguinal lymph nodes are a part of the

Fig. 7 (case 1).—Section of serosal portion of the endometrial carcinoma of the transverse colon. Note the numerous cystic spaces, containing granular material. The large cyst in the center shows papillary masses of adenocarcinoma. To the extreme right, just above the circular muscle layer of the colon, is a subserosal artery, completely plugged with carcinoma cells. To the extreme left (arrow) is a cyst surrounded by a narrow zone of lymphocytes. It is in the lining of this cyst that cilia were found (fig. 9). Very low power.

30. Schindler, R.: Statistische und anatomische Ergebnisse bei der Freund-Wertheimschen Radikaloperation des Uterus-Karzinoms, Monatschr. f. Geburtsh. u. Gynäk. **23**:502, 1906.

31. Sampson, J. A.: Intestinal Adenomas of Endometrial Type: Their Importance and Their Relation to Ovarian Hematomas of Endometrial Type (Perforating Hemorrhagic Cysts of the Ovary), Arch. Surg. **5**:217 (Sept.) 1922.

32. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 600.

lymphatic drainage of the uterus. Meyer [33] also observed both polypoid proliferation into lymph channels of the mesocolon and epithelial inclusions in a lymph node in a case of endometriosis of the sigmoid flexure. The presence of endometrial-like inclusions in the inguinal lymph node

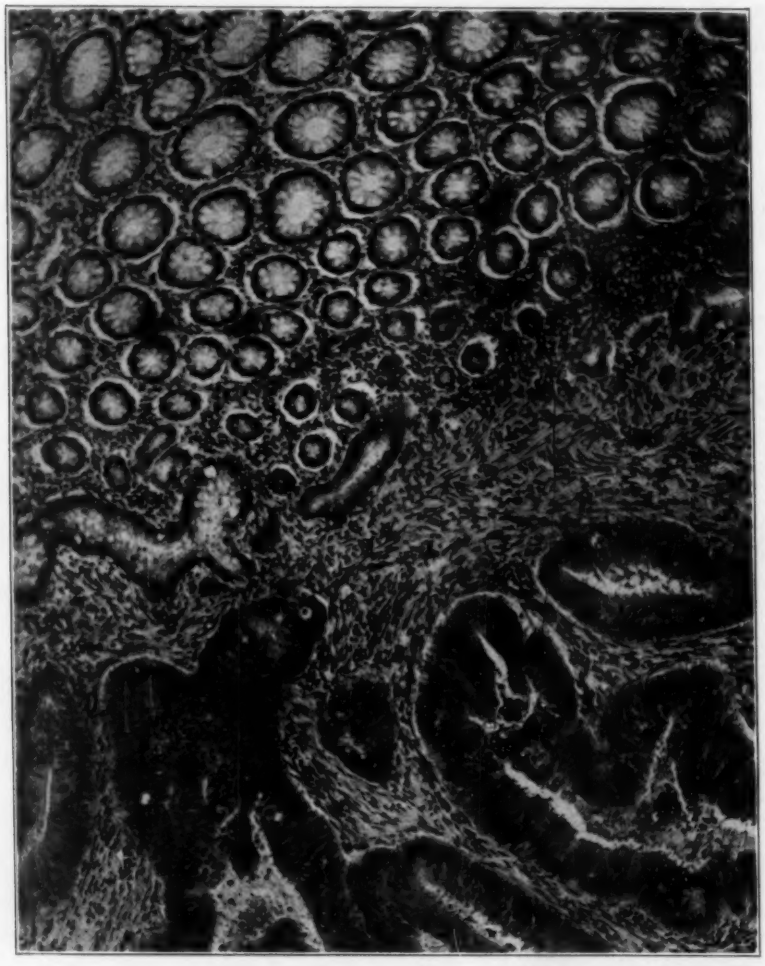

Fig. 8 (case 1).—Transverse colon. Typical carcinoma cells, invading the intestinal mucosa. High power.

and of adenomyoma of the endometrial type in the groin in case 1 strongly points to a lymphatic dissemination of particles of endometrial tissue.

33. Meyer, R.: Ueber entzuendliche heterotope Epithelwucherungen im weiblichen Genitalgebiete und ueber eine bis in die Wurzel des Mesocolon ausgedehnte benigne Wucherung des Darmepithels, Verhandl. Deutsch. Path. Ges. in Kiel, 1908; Virchows Arch. f. path. Anat. **195**:487, 1909.

Meyer,[33] in 1908, was the first to report a case of intestinal endometrial adenomyoma in the sigmoid flexure. In 1913, de Josselin de Jong [34] described a case of endometriosis of the small intestine (ileum). In 1926, Polster collected eighty cases of intestinal endometriosis from the literature. In Sampson's [31] experience, intestinal adenoma of the endometrial type occurs in more than one half of the cases of ectopic endometrial adenomas. He has found them most frequently on those portions of the intestinal tract which are usually found in the pelvis. In case 1, the tumor occurred in the midtransverse colon and is unique in that it occurred above the level of the umbilicus and that it became carcinomatous. It is much simpler and pathologically sounder to regard the observations in this case as evidence of one process than to consider them separate different entities.

Fig. 9 (case 1).—Cyst in wall of transverse colon. Cilia in the lining of a cyst, mentioned in figure 7. High magnification.

That malignant degeneration can occur in an ectopic endometrial growth is shown in this endometrial carcinoma of the transverse colon. In 1882, Babes [35] described a case of carcinomatous degeneration of a uterine adenomyoma. Other observers like von Recklinghausen, Rolley,[36] Dillmann,[37] Meyer, Schwab,[38] Cullen,[39] Kaufmann,[40] Polano,[41]

34. De Josselin de Jong, R.: Subseroese Adenomyomatose des Duenndarms, Virchows Arch. f. path. Anat. **211**:141, 1913; Frankf. Ztschr. f. path. **22**:400, 1919-1920.

35. Babes, V.: Ueber epitheliale Geschwuelste in Uterusmyomen, Centralbl. f. Gynäk. **24**:382, 1882.

36. Rolley: Ueber einen Fall von Adenomyoma uteri mit Uebergang in Carcinom und Metastasenbildung, Virchows Arch. f. path. Anat. **150**:555, 1897.

37. Dillmann, H.: Adenomyome des Uterus und ihre Beziehungen zum Krebs, Ztschr. f. Krebsforsch. **2**:333, 1904.

Moench,[42] Becker [43] and Lindsay [44] reported similar observations. Schwab's case is interesting in that three identical carcinomatous changes were present simultaneously in three separate and distinct adenomyomata uteri. In Bauereisen,[45] Kaufmann [40] and Thomson's [46] cases of uterine adenomyoma, a sarcomatous degeneration was present. Iwanoff [8] had observed a case of both a sarcomatous and a carcinomatous degeneration in an adenomyoma of the uterine wall. Metastases to various regions as lymph nodes, liver, lung, intestine, spleen, omentum and bones were variously reported by Rolley, Dillmann and Thomson. In case 1, as mentioned previously, metastases were present in the mesenteric lymph nodes, omentum and liver. As regards malignant transformation of ectopic endometriosis, besides our own, we have been able to find only one record; in seven of sixteen specimens of ovarian carcinomas without evidence of cancer of the uterus Sampson [1] found benign endometrial-like tissue in the ovary bearing the same histologic relationship to malignant growths as benign uterine mucosa bears to adenocarcinoma arising in the body of the uterus.

CASE 2.—*Endometriosis in the Vermiform Appendix, Associated with Endometriosis of the Fallopian Tube.*

S. D., aged 33, married, primipara, about one week before operation complained of soreness and pain in the lower part of the abdomen after eating. There was nausea, but no vomiting. The past history showed that there was intense pain over the appendix during the menses. At operation, a bilateral tubo-ovarian mass and the appendix were removed.

Grossly, the larger tubo-ovarian mass, 6.5 by 4.5 by 3 cm., presented a reddish-gray roughened outer surface with large masses of fibrin attached. The wall of the fallopian tube was enormously thickened, from 5 to 6 mm., and bluish-gray. Two small thin-walled cysts bulged from the reddened serosa of this tube. The mucosa was thick and reddish gray. The lumen contained a small amount of pus.

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38. Schwab, M.: Multiple Adenomyomata uteri in karzinomatoeser Degeneration, Beitr. z. Geburtsh. u. Gynäk. **12**:102, 1907.

39. Cullen, T. S.: Adenomyoma of the Uterus, Philadelphia, W. B. Saunders Company, 1908, chapter 19.

40. Kaufmann, E.: Lehrbuch der speziellen pathologischen Anatomie, ed. 5, Berlin, G. Reimer, 1909, p. 986.

41. Polano, O.: Zur Pathologie des Uterus, Ztschr. f. Geburtsh. u. Gynäk. **67**: 413, 1910.

42. Moench, G. L.: Zur Pathologie des Carzinoms, Ztschr. f. Geburtsh. u. Gynäk. **80**:1, 1917-1918.

43. Becker, C.: Karzinomatoese Degeneration heterotoper Epitheleinschluesse im Uterus, Zentrabl. f. Gynäk. **49**:2333, 1925.

44. Lindsay, W. S.: Variations in the Prognosis of Endometrial Carcinoma as indicated by the Histological Structure, Surg. Gynec. Obst. **43**:1, 1927.

45. Bauereisen, A.: Ein bermerkenswerter Fall von Adenomyoma uteri sarcomatosum, Hegar's Beitr. z. Geburtsh. u. Gynäk. **9**:313, 1904-1905.

46. Thomson, A. P.: Malignant Endometrioma with Metastases in the Lungs, Proc. Roy. Soc. Med. **19**:16, 1925-1926.

The smaller tubo-ovarian mass, more irregular in form, showed the outline of a swollen tube, 9.5 cm. long and 1.5 cm. in its largest diameter. A small amount of ragged ovarian tissue was firmly united to the tube and the fimbriated end was closed. The lumen contained a small amount of turbid yellowish fluid. The appendix, 4 by 0.7 cm., had a roughened serosa with injected vessels. The lumen was patent except the terminal centimeter.

The microscopic examination showed a bilateral chronic salpingo-oöphoritis. The two small thin-walled cysts of the tubal serosa were lined by a single wavy layer of cuboidal ciliated cells, and in the adhesions between the tube and the ovary there were many isolated lumina, lined by similar ciliated epithelium surrounded by muscle fibers. In the ovary itself, there was no demonstrable endometriosis.

The wall of the appendix was markedly thickened, notably the serosa in which the newly formed fibrous tissue was infiltrated with lymphocytes. In one area,

Fig. 10 (case 2).—Endometriosis of the appendix. In the thickened serosa and outer portion of the muscularis is a collection of gland tubules, surrounded by a narrow zone of cellular stroma. To the extreme left are the lymph follicles of the mucosa. Low power.

in both the subserosa and outer portion of the muscularis, there was a collection of dilated gland tubules, lined by a single layer of ciliated columnar cells. Each tubule was surrounded by a thin zone of cellular stroma (fig. 10). The subserosa near the tip was composed of thick, injected and hemorrhagic granulation tissue and contained two small gland tubules, lined by a single layer of cuboidal epithelium with cilia.

Case 3.—*Endometriosis in the Vermiform Appendix.*

J. G. M., aged 22, single, for the past year had had mild attacks of pain in the right lower quadrant, more severe during menstruation, which was regular. Appendectomy was performed. The uterus and adnexa were normal.

The appendix was 7 cm. long with an average diameter of 8 mm. The tip was firm and nodular and the cut surface showed some gray areas in the

thickened and injected serosa. The mucosa was redundant. On microscopic examination, the wall including the serosa was seen to show a marked scarlike fibrosis with hyalinization, and in the thickened serosa near the tip were islands of glandular structures surrounded by a cellular stroma. These glands were lined by a wavy layer of high cylindric cells. Cilia were not demonstrable. Some of the gland lumina were distended with a hemorrhagic fluid (fig. 11). The stroma itself was markedly injected, ecchymotic and contained considerable numbers of endothelial leukocytes with engulfed blood pigment, evidence of old hemorrhage.

Comment.—Both of these patients presented symptoms of appendical colic, definitely related to the menstrual periods. Endometriosis of the appendix has been observed by Hueter,[47] Sampson,[48] Meigs,[49] Dougal,[50] Pick,[51] Suzuki,[52] Outerbridge,[53] Seelig[54] and Schochet.[55] Meig's patient was operated on for acute appendicitis, and an inflamed appendix bound down by dense adhesions was found. One of Sampson's patients complained of two recent attacks of appendicitis. In Schochet's case, there was a dull aching pain in the right lower quadrant with symptoms of chronic appendicitis. As a rule, the associated pelvic pathologic process masked the appendical involvement. In Dougal's case and in our two cases, the pains were more severe during the menses. In case 2, the appendical symptoms were complicated by those of tubo-ovarian disease, while in case 3, the only symptoms were those of chronic appendicitis. The pathogenesis of these ectopic proliferations may be explained as a metaplasia of embedded serosa when evidence of a chronic inflammation is present, or as a direct extension in adhesions to an endometrioma of the adnexa, or they may be ascribed to implantation when there is an associated pelvic endometriosis.

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47. Hueter, C.: Ueber entzuendliche Druesenartige Neubildungen des Peritoneums (Peritonitis adenoides), Frankf. Ztschr. f. Path. **21**:283, 1918.

48. Sampson, J. A.: Inguinal Endometriosis, Am. J. Obst. Gynec. **10**:462, 1925; footnotes 1, 5 and 31.

49. Meigs, J. V.: Endometrial Hematomas of the Ovary, Boston M. & S. J. **187**:1, 1922; Adenomyomata or Tumors Composed of Endometrial-Like Tissue, ibid. **195**:343, 1926.

50. Dougal, D.: Andenomyoma Involving the Vermiform Appendix, J. Obst. & Gynec. Brit. Emp. **30**:224, 1923.

51. Pick, L.: Ueber endometrioide Wucherungen an der Appendix, Verhandl. Aerztlicher Berl. Gess. f. path. Anat. u. vergl. Path. Klin. Wchnschr. **3**:502, 1924.

52. Suzuki, S.: Ueber endometrioide Adenomyom und endometrioide Adenomatose des Wurmfortsatzes, Virchows Arch. f. path. Anat. **250**:579, 1924.

53. Outerbridge, G. W.: Cystic Lesions of possible Endometrial Origin in the Appendix, Am. J. Obst. & Gynec. **10**:545, 1925.

54. Seelig, M. G.: Endometrial Adenoma (Implantation) in the Vermiform Appendix, Am. J. Obst. & Gynec. **11**:461, 1926.

55. Schochet, S.: So-Called Primary Adenocarcinoma of the Appendix, Tr. Chic. Gynec. Soc., Jan. 21, 1927; Am. J. Obst. & Gynec. **14**:684, 1927.

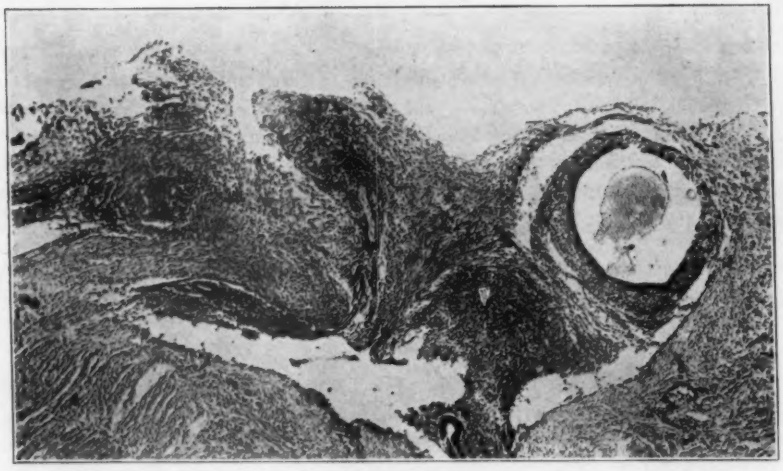

Fig. 11 (case 3).—Endometriosis of the appendix. On the surface is a large amount of cellular stroma, in which three gland tubules are embedded. The large circular dilated tubule contains red blood cells. The dense hazy appearance of the stroma is due to hemorrhage. Medium power.

Fig. 12 (case 4).—Endometriosis in a laparotomy scar. The dark line at the center is a fold in the section. At the upper left hand corner is the epidermis. The scar tissue beneath is literally riddled with irregular glandular spaces of various sizes. The larger spaces contain desquamated cells and blood. In the lower right hand corner is a typical pseudoglomerulus of von Recklinghausen (arrow). Very low power.

CASE 4.—*Endometriosis in a Laparotomy Scar.*

E. H., aged 40, unmarried, never pregnant, had had an appendectomy performed when 11 years of age. Twenty-six and one-half years after the operation, a lump had appeared in the old appendectomy scar. The lump had not occasioned any distress until six months before the patient was admitted to the hospital, at which time it became painful for one day. The menses were normal in every respect. Operation revealed a postoperative hernia in the wall of which there was an ill-defined tumor mass. This mass measured 35 by 20 mm. and grossly was dense with gray and red markings. It was incorporated with the deeper layers of the skin and was not demarcated from the surrounding fat.

Fig. 13 (case 4).—Same section as in figure 12. This cystic space was present near the epidermal surface. Observe the wavy epithelial lining. The contents consist of fibrin and the formed elements of the blood. In the left lower corner is a portion of a large island of characteristic cellular stroma (arrow). Medium power.

The microscopic sections showed islands of endometrial-like growth in the tumor mass in the scar tissue (fig. 12). The gland tubules were lined by columnar or low cuboidal cells, many of which were ciliated. Many glands were dilated, and the lining epithelium presented a wavy surface like the glands seen in the menstrual endometrium (fig. 13). The lumina contained fibrin, numerous red blood cells or other cellular debris. Some of these islands of endometrial tissue were evidently the pseudoglomerular structure of von Recklinghausen (fig. 14). The stroma about these glomeruli was infiltriated with red blood cells, small lymphocytes,

plasma cells and occasional polymorphonuclear leukocytes and contained large oval cells like the stroma cells of the uterine mucosa. A foreign body giant cell reaction around cotton threads was present in the immediate vicinity of the endometrial islands. In sections stained by Perl's method, iron pigment was most abundant in and around the endometrial islands. The van Gieson stain demonstrated smooth muscle fibers.

Comment.—In 1902, Meyer [56] reported the first case of endometriosis in a laparotomy scar. To date, eighty-seven cases are on record including our own (table 1). Almost all of these occurred between the ages of 20 and 45 years, with only two cases after 45 years,

Fig. 14 (case 4).—Same section as in figure 12. The pseudoglomerular structure of von Recklinghausen with endometrial tubules surrounded by a cellular stroma is well shown. Medium power.

one at 46 and the other at 50 years. Eight cases were in single women and twenty-seven in married women. There were thirty-one cases following ventrofixation, twenty-eight after salpingectomy, oöphorectomy, or both, nineteen after cesarean section, fifteen after appendectomy, five after hysterectomy and the remainder after miscellaneous operations on the pelvic organs. The swelling or tumor made its

56. Meyer, R.: Ueber eine adenomatoese Wucherung der Serosa in einer Bauchnarbe, Verhandl. d. Ges. f. Geburtsh. u. Gynaek., zu Berlin, Dec. 12, 1902; Ztschr. f. Geburtsh. u. Gynäk. **49**:32, 1903.

TABLE 1.—*Collected Cases of Endometriosis Following Operations*

No.	Author	Age	Marital Pregnancies	Previous Operation	Occurrence After Operation	Location of Growth in Scar	Internal Adhesions Between	Symptomatology	Effect of Menses	Menses
1	Meyer: Ztschr. f. Geburtsh. u Gynäk. **49**: 32, 1903	35	.....	Ventrofixation; salpingectomy	.....	Below navel	Uterus, omentum and scar	Painful node in scar	.....	.....
2	Amos: Zentralbl f. Gynäk. **29**: 145, 1905	45	Married 1 pregnancy	Ventrofixation	Soon after	.....	.....	Two painful nodes in scar	.....	.....
3	Stratz: Zentralbl. f. Gynäk. **29**: 1303, 1905	44	Married nullipara	Ventrofixation; oöphorectomy; myomectomy	3½ years	.....	.....	Bluish-red lump painful and bleeds on touching; recurrence 4 months after removal of above lump	....	Regular
4	Opitz: Verhandl. der deutsche ges. f. Gynaek., 1911, 14 Congress, Muenchen, p. 567-570	..	.....	Operation for adnexal disease	.....	.....	Uterus, omentum and scar	Severe pain in old scar	.....	.....
5	Klages: Ztschr. f. Gebertsh. u. Gynäk. **70**: 858, 1912	Mid. dle-aged	.....	Ventrofixation; salpingo-oöphorectomy; appendectomy	4 years	Lower end	Uterus, omentum and scar	Very painful and sensitive lump; prickling and pressure sensations	Marked reddening	.....
6	Amann: Monatschr. f. Geburtsh. u. Gynäk. **42**: 492, 1915	..	.....	Removal of benign ovarian tumor	Some years	.....	.....	.....	.....	.....
7	v. Franque: Zentralbl. f. Gynäk. **40**: 953, 1916	39	Married 5 pregnancies	Repair of perforated uterus caused by abortion; salpingectomy	Short time	Lower end	.....	Lately small node growing more quickly	.. ..	.....
8	Meyer: Zentralbl. f. Gynäk. **43**: 745, 1919	..	.....	Ventrofixation 8 years ago	.....	.....	.....	.....	....	.....
9	Fraas: Zentralbl. f. Gynäk. **43**: 750, 1919	45	Married 1 pregnancy	Ventrofixation 20 years ago	.....	Lower half	Uterus and scar	.....	....	.....
10	Mahle and MacCarty: J. Lab. & Clin. Med. **5**: 218, 1920; Lemon and Mahle: Arch. Surg. **10**: 150 (Jan.) 1925; M. Clin. N. Amer. **1**: 1125 1925	46	Married 9 pregnancies	Ventrofixation	Few years	Middle	Uterus and scar	Hard mass gradually growing for one year	Painful following periods	.....
11	Idem.	30	.....	Ligaments shortened	3 years	Lower end	Tube and scar	Painful to touch	Painful following menses	.....

12	Idem.	50	Single	Appendical abscess drained	21 years	.....	.....	Small nodes in scar gradually growing	Painful at periods	Normal
13	Cullen: Arch. Surg. **1**: 215, 1920	34	Married 2 pregnancies	Repair of perforated uterus from abortion	9½ years	Lower end	.....	Tenderness; increase in size of lump	.....	.....
14	Cullen and Burnam: Am. J. Obst. & Gynec. **4**: 562, 1922	..	.....	Extensive operation for adenomyoma of uterus and of recto-vaginal septum	8 years	.....	.....	.....	.....	.....
15	Idem.	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
16	Idem.	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
17	Sampson and Mallory: Tr. Am. Gynec. Soc. **47**: 56, 1922; Am. J. Obst. & Gynec. **4**: 451, 1922	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
18	Idem.	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
19	Dietrich: Zentralbl. f. Gynäk. **47**: 1055, 1923	..	.....	Ventrofixation	.....	.....	.....	.....	.....	.....
20	Bergmann: cited by Dietrich	..	.....	.....	.....	.....	.....	.....	.....	.....
21	Hedinger: Schweiz. med. Wchnschr. **4**: 828, 1923	30	.....	Appendectomy 8 years ago	.....	.....	.....	Fecal fistula	.....	.....
22	Lochrane: J. Obst. & Gynec. Brit. Emp. **30**: 213, 1923	38	Married 2 pregnancies	Ventrosuspension	3 years	.....	Uterus and scar	Mass in scar gradually enlarged	.....	Menorrhagia; dysmenorrhea
23	Tobler: Frankfurt Ztschr. f. Path. **29**: 558, 1923	22	.....	.....	.....	.....	.....	.....	.....	.....
24	Idem.	25	.....	Appendectomy 11 years ago; tubal sterilization 9 years ago	½ year after second operation	Upper end	None	Hazel-nut sized lump in scar	Swelling and pain during periods	.....
25	Idem.	41	Married 1 pregnancy	Tubal pregnancy	10 years	Middle	None	Small hernia in scar; walnut-sized lump	Swelling and pain during periods	.....
26	Idem.	32	.....	Interruption of pregnancy; tubal sterilization	6 months	.....	None	Small lump	Pain	.....
27	Idem.	30	.....	Appendectomy with suppuration of wound	Very soon after	.....	.....	Small lump	.....	.....

TABLE 1.—*Collected Cases of Endometriosis Following Operation—Continued*

No.	Author	Age	Marital Pregnancies	Previous Operation	Occurrence After Operation	Location of Growth in Scar	Internal Adhesions Between	Symptomatology	Effect of Menses	Menses
28	Lauche: Virchows Arch. f. path. Anat. **243**: 208, 1923	30	Married 1 pregnancy	Ventrofixation; bilateral salpingectomy	Soon after	Lower end	.....	Walnut-sized mass	Cysts filled with blood during menses then subsided	.....
29	Idem.	35	Married 1 pregnancy	Ventrofixation; oöphorectomy	1 month	.....	Uterus and scar	Pain and swelling; recurrence 1 year later	Pain before and during periods with swelling	Dysmenorrhea
30	Idem.	37	Married 3 pregnancies, several abortions	Salpingo-oöphorectomy	7 years	Lower end	Stump of tube and scar	Pain more in last year	.....	Dysmenorrhea, menorrhagia
31	Idem.	26	Single	Ventrofixation; removal of ovarian tumor	6 years	Lower part	Uterus and scar	No complaint between periods	Pain before and after periods with swelling	Always regular
32	Petitpierre: cited by Lauche	..	.....	.....	.....	.....	.....	.....	.....	.....
33	Pick:	..	.....	.....	.....	.....	.....	.....	.....	.....
34	Vassmer: Arch. f. Gynäk. **123**: 187, 1924-1925	24	.....	Ventrofixation	6 months	.....	.....	Walnut-sized mass in scar; recurrence after removal 3 months later	Painful during periods with bloody discharge	Dysmenorrhea
35	Sampson: Surg. Gynec. Obst. **38**: 287, 1924	30	.....	Cesarean; salpingectomy for tubal pregnancy 2 years later; hysterectomy 4 years later	Few weeks after last operation	.....	.....	Gradual increase in size of scar tumor; no pain; no discomfort	.....	.....
36	Abbott: Boston M. & S. J. **191**: 1159, 1924	34	Married 1 pregnancy, 2 abortions	Appendectomy 7 years ago; cesarean, 3½ years ago; therapeutic abortion twice	3 months after cesarean	Middle	.....	Slightly tender lump gradually increased in size	.....	Normal
37	Idem.	37	Married	Cesarean	7½ years	Upper part above navel	.....	Slight soreness at times	.....	.....

38	Mandl: Zentralbl. f. Gynäk. **49**: 664, 1925	26	Married 1 pregnancy	Appendectomy	9 years	.....	None	Small thickening gradually grew larger; sensitive to pressure; bluish cyst	Painful and more sensitive before and during periods; dark bloody discharge	Normal
39	Lemon and Mahle: Arch. Surg. **10**: 150 (Jan.) 1925; Med. Clin. N. Amer. **1**: 1125, 1925	35	.....	Ventrofixation 4 years ago	.....	.....	Uterus and scar	Tenderness in lower part of abdomen	Especially tender during periods	.....
40	Idem.	38	.....	Alexander operation 4 years ago	.....	.....	Uterus and scar	Soreness in pelvis	Symptoms worse during periods	.....
41	Idem.	43	.....	Ventrosuspension 12 and 4 years ago	1 year after second operation	.....	Uterus and scar	Tingling senstaion and slight pain in scar	Always worse before and during periods	.....
42	Idem.	27	.....	Salpingectomy 4 years ago; operation for adhesions 2 years ago	.....	.....	Uterus and scar	Tender mass	.....	.....
43	Idem.	36	.....	Curettage and ventrosuspension 3 years ago	6 weeks	Lower end	Uterus and scar	Slightly tender; intermittently discharged serosanguinous fluid; gradually became larger	.....	.....
44	Idem.	40	.....	Oöphorectomy and hysterectomy 6 years ago	Recently noted mass	.....	.....	Mass in scar	.....	.....
45	Idem.	35	.....	Bilateral salpingectomy; right oöphorectomy and appendectomy 9 years ago	6 years	Lower part	Omentum and scar	Mass in scar	Pain and larger during periods	.....
46	Rosenstein: Monatschr. f. Geburtsh. u. Gynäk. **49**: 654, 1925; Zentralbl. f. Gynäk. **49**: 1512, 1925	23	.....	Ventrofixation; salpingectomy	5 years	Middle	.....	Tender, painful mass	.....	Regular
47	Luetzenkirchen: Zentralbl. f. Gynäk. **49**: 1208, 1925	..	.....	Cesarean intestinal resection	.....	.....	.....	Fistula	.....	.....
48	Keller: Monatsch. f. Geburtsh. u. Gynäk. **70**: 110, 1925	..	.....	.....	.....	.....	.....	.....	.....	.....

TABLE 1.—*Collected Cases of Endometriosis Following Operation—Continued*

No.	Author	Age	Marital Pregnancies	Previous Operation	Occurrence After Operation	Location of Growth in Scar	Internal Adhesions Between	Symptomatology	Effect of Menses	Menses
49	Gerlach: Verhandl. der. deutsch. path. ges., 1925, p. 431	..	.....	Ventrofixation	.....	.....	.....	Thread suppuration and fistula	Bloody discharge from fistula which closed in the interval	.....
50	Pankow: Monatschr. f. Geburtsh. u. Gynäk. **71**: 361, 1925	40	.....	Appendectomy when a child	Many years	.....	.....	Gradually increasing mass; fistula	Bloody discharge from fistula during each period	.....
51	Heaney: Am. J. Obst. & Gynec. **10**: 625, 1925	32	2 abortions	Cesarean	1 year	Upper end	.....	Sore, tender lump	Swelling and pain during periods	.....
52	Idem.	38	5 pregnancies, 3 miscarriages	Ventrosuspension; tubal sterilization; fundus opened to remove embryo	Soon after	Right end of transverse scar	.....	Tender swelling	Very painful and swollen during periods	.....
53	Danforth: Am. J. Obst. & Gynec. **10**: 630, 1925	20	1 pregnancy	Cesarean; tubal sterilization	2 years	Lower third	.....	Tender hard mass	None	.....
54	Graves: Am. J. Obst. & Gynec. **10**: 665, 1925	32	.....	Multiple myomectomy; ventrosuspension	Soon after	One end of scar	.....	Hard mass slowly increasing in size; recurrence 3 years after its removal	Bloody discharge from infected wound during period	Dysmenorrhea
55	Halban: Arch. f. Gynäk. **124**: 457, 1925	20	Single 1 abortion	Ventrofixation; partial oöphorectomy	6 years	Left end of transverse scar	.....	Sensitive lump in scar	Swelling and pain during periods	Always irregular; metrorrhagia; dysmenorrhea
56	Mark: Med. Klin. **20**: 754, 1925; Sternberg: Biol. u. path. des. Weibes, 1925, vol. 5, p. 720	28	.....	Appendectomy; suppuration and drainage	11 years	.....	Pelvic organs and fistula	Fistula	Bloody discharge during periods	.....
57	Meyer: cited by Polster: Virchows Arch. f. path. Anat. **259**: 96, 1926	..	.....	Cesarean	.....	.....	.....	.....	....	.....
58	Idem.	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
59	TeLinde: Northwest Med. **25**: 123, 1926	32	.....	Hysterectomy 2 years ago	1 year	Lower end	.....	Small tender lump which gradually increased in size; painful	Pain worse during periods	.....

60	Nicholson: Guy's Hosp. Rep. **76**: 188, 1926; J. Obst. & Gynec. Brit. Emp. **33**: 620, 1926	36	Single	Colotomy; pelvic suppuration 2 years ago	.....	Lower end	.....	Mass in scar	Pain and swelling	.....
61	Novak: Am. J. Obst. & Gynec. **12**: 484, 1926	..	.....	Salpingo-oöphorectomy with drainage 5 years ago	.....	.....	.....	Very tender; no increase in size	None	.....
62	Neuweiler: Schweiz. Med. Wchnschr. **7**: 545, 1926	34	.....	Ventrofixation	Soon after	.....	Uterus and scar	Mass in scar	Swelling and marked pain during and after periods	.....
63	Idem.	30	.....	Removal of ovarian dermoid; hysterotomy	2 years	Middle	.....	Swelling in scar lately very sensitive	None	.....
64	Jeanneret: Rev. med. de la Suisse Romande **46**: 836, 1926	27	Single	Appendectomy; partial oöphorectomy; ligaments shortened	3 years	On left side 4 cm. from scar	.....	Painful mass in scar	Pain very intense during periods	Dysmenorrhea
65	Berkeley: J. Obst. & Gynec. Brit. Emp. **33**: 657, 1926	30	Married 1 pregnancy	Cesarean	3 years	Lower end	.....	Sensitive painful mass gradually increased in size	More painful during periods	Normal
66	Bonney: J. Obst. & Gynec. Brit. Emp. **33**: 657, 1926	29	Married	Hysterotomy	1 year	Middle of transverse scar	.....	Mass in scar	Pain and temporary increase in size during periods	.....
67	Polster: Virchows Arch. f. path. Anat. **259**: 96, 1926	36	Married nullipara	Ventrofixation 5 years ago	Sometime after	.....	Tube and scar	Mass in scar	Pain during period	Dysmenorrhea
68	Walz: Centralbl. f. allg. Path. **37**: 290, 1926	32	Married 1 pregnancy, 1 abortion	Ventrofixation; oöphorectomy	1 year	.....	.....	Mass in scar	Pain in scar before and after periods	.....
69	Idem.	30	Single	Ventrofixation 3 years ago	.....	.....	.....	Painful mass in scar	Pain more marked during periods	.....

TABLE 1.—*Collected Cases of Endometriosis Following Operation—Continued*

No.	Author	Age	Marital Pregnancies	Previous Operation	Occurrence After Operation	Location of Growth in Scar	Internal Adhesions Between	Symptomatology	Effect of Menses	Menses
70	Idem.	33	Married	Ruptured tubal pregnancy 9 years ago	.....	.....	.....	Mass in scar; fistula	Bloody discharge from fistula during period	.....
71	Schwarz: Am. J. Obst. & Gynec. **13**: 331, 1927	28	.....	Cesarean	2 years	Upper end	.....	Sensitive spot in upper end of scar	... .	.....
72	Maes: Am. J. Surg. **2**: 539, 1927	38	.....	Hysterectomy	Soon after	Whole scar	.....	Swollen scar	Severe pain and swelling during periods with bloody discharge	.....
73	Idem.	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
74	Idem.	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
75	Polano: Zentralbl. f. Gynäk. **51**: 963, 1927	45	Married 7 pregnancies	Tubal pregnancy	1 year	Upper part	Uterus and scar	Pain and marked swelling around plum-sized vesical	Bloody discharge from vesical	Regular
76	Pratt: J. Michigan M. Soc. **26**: 82, 1927	45	Single	Curettage ventrofixation; hysterectomy	Soon after last operation	.....	Uterus and scar	Tender and painful mass in scar	Pain intense	Dysmenorrhea
77	Idem.	37	Married nullipara	Ventrofixation 10 years ago	.....	.....	Uterus and scar	Tender scar	Pain, swelling and bloody discharge	.....
78	Idem.	45	Married	Appendectomy; suspension	6 years	.....	.....	Small lump in scar	Burning and pain in lump before and during period	.....
79	Idem.	41	.....	Appendectomy 17 years ago; suspension 16 years ago; curettage 7 years ago	1 year after third operation	Lower end	.....	Tender; painful lump	More severe little dark discharge from lump	.....

80	Ballin: Surg. Gynec. Obst. **46**: 525, 1928	27	.....	Salpingo-oöphorectomy 6 years ago	.....	.....	Uterus and scar	Bloody discharge from fistula	More painful and increased discharge; fistula had to be opened several times to relieve painful retention	.....
81	Idem.	35	Married nullipara, 1 miscarriage	Removal of uterine tumor and salpingo-oöphorectomy; 4 years ago; drainage	.....	Lower end	Uterus, omentum and scar	Tender mass slowly growing	Very painful with dark bloody discharge	Menorrhagia
82	Idem.	22	.....	Oöphorectomy 6 years ago	.....	Lower end	Cystic ovary and scar	Hard mass in scar requiring lancing or broke down to discharge blood	Always painful during period; relief on opening one of the blood cysts	.....
83	Williams: Tr. N. Y. Obst. Soc., May, 1928	..	.....	Cesarean	.....	.....	.....	.....	.....	.....
84	Douglass: J. A. M. A. **90**: 1858 (June 9) 1928	41	.....	Appendectomy with peritonitis and drainage 11 years ago; fixation 8 years ago	Few years	Lower end	.....	Painful defecation; painful scar	Worse before and during period	.....
85	Idem.	20	Married	Salpingectomy and suspension 2 years ago; abscess in scar and drainage 1½ years ago	.....	.....	Multiple pelvic adhesions	Painful lump	.....	.....
86	Idem.	..	.....	Miniature cesarean	6 months	Upper end	.....	Lump in scar	Marked tenderness before and during periods	.....
87	Hosoi and Meeker: In this article	40	Single	Appendectomy	26½ years	Lower end	.....	Lump in scar; painful for one day only	None	Normal

appearance soon after operation, or as in our case, as late as twenty-six and one-half years afterward. The lower end of the operative scar appears to be the site of predilection. Internal adhesions to the pelvic organs or omentum are noted in thirty cases. The chief signs or symptoms are swelling, tenderness and pain, all increased before, during or after the menstrual period. Menses tend to be abnormal with dysmenorrhea or menorrhagia. Either Ballin's "menstrual fistula" or a broken-down blood-filled cyst occurred in fifteen cases. Smooth muscle fibers, lime deposits, blood, iron pigment and suture material have been found, as well as the endometrial stroma and gland tubules with cilia.

As regards the pathogenesis of endometrium in laparotomy scars, the transplantation theory would seem to explain the majority of the cases, as most occurred after operations on the uterus and adnexa. In Meyer's case, small glands and cysts were especially numerous near silk-worm threads, and for this reason, the suture needle has been held responsible in cases in which a ventrofixation of the uterus has been performed. In one of Neuweiler's [57] cases, an endometriosis developed in a perineal scar occasioned by a pregnancy five years previous. Experimental proof of the transplantability of endometrium from the uterus to the abdominal organs has been fully shown by Jacobson,[58] Albrecht,[59] Bell,[60] Dossena,[61] Stilling,[62] Ferracciu,[63] Katz and Szenes,[64] Traut,[65] O'Keefe and Crossen.[66] Cron and Gey [67] were able to grow human endometrium in

57. Neuweiler, W.: Beitrag zur Klinik der endometroiden Wucherungen, Schweiz. med. Wchnschr. **7**:545, 1926.

58. Jacobson, V. C.: The Auto-Transplantation of Endometrial Tissue in the Rabbit, Arch. Surg. **5**:281 (Sept.) 1922; Further Studies in Auto-Transplantation of Endometrial Tissue in the Rabbit, Am. J. Obst. & Gynec. **6**:257, 1923.

59. Albrecht: Diskussion zu den Vortraegen der Herren Vogt und Katz und Szenes, Bäyerischer Ges. f. Geburtsh. u. Gynaek., Muenchener Gynaek. Ges., Zentralbl. f. Gynäk. **48**:2402, 1924.

60. Bell, B., cited by Jacobson, V. C. (footnote 58, first reference).

61. Dossena, G.: Neoformazioni sperimentali da autotrapianto di mucosa uterina, Tumori **12**:291, 1926.

62. Stilling, H.: Versuche ueber Transplantation IV Mitteilung. Das Ergebnis der Transplantation von Uterusgewebe in die Milz, Beitr. z. path. Anat. **47**:499, 1909-1910.

63. Ferracciu, D.: Sulla producione sperimentali di endometriomi, Riv. ital. di ginec. **4**:235, 1926.

64. Katz, H., and Szenes, A.: Untersuchungen ueber die Verplanzung des Endometriums in die peritonealhoehle beim Kaninchen, Zentralbl. f. Gynäk. **48**: 2400, 1924; Ztschr. f. Geburtsh. u. Gynäk. **90**:74, 1926.

65. Traut, H., cited by Novak, E.: Tr. Am. Gynec. Soc., 1927, Am. J. Obst. & Gynec. **15**:101, 1928.

66. O'Keefe, D., and Crossen, R. J.: The Auto-Transplantation of Endometrial Tissue in Dogs, J. Missouri M. A. **24**:252, 1927.

67. Cron, R. S., and Gey, G.: Viability of Cast-Off Menstrual Endometrium, Am. J. Obst. & Gynec. **13**:645, 1927.

tissue cultures. They used the fragments of epithelium (decidua menstrualis) normally found in the menstrual flow and obtained viable cultures. They did not curet the uterus. In our case, endometrial tissue from a possible pelvic ectopic endometrioma may have become implanted in the hernia sac. Unfortunately, no exploration of the abdominal cavity was undertaken. On the basis of a chronic inflammation of twenty-six and one-half years' duration as evidenced by the foreign body reaction, the metaplasia theory appears more applicable.

The serosal metaplasia theory and the lymphatic theory have been applied to those instances in which a simple appendectomy has been performed and in which there was no hernia. Tobler, by serial sections, demonstrated a connection between the ectopic endometrial tissue and the peritoneal serosa, and Dietrich showed the development of glomerular-like structures from the serosa. In these cases in which the fundus uteri was adherent to the tumor mass in the laparotomy scar, serial sections could demonstrate no endometrial tubules in the adherent portion of the uterus. This shows that the laparotomy tumors in the literature were not due to an extension from the uterine cavity. Ewing [68] believed that no one theory can explain all the cases.

As regards the origin of the cellular stroma and the smooth muscle fibers in cases of adenomyoma, Meyer, Renisch,[69] Nicholson [70] and others expressed the opinion that they are derived from the tissues of the new habitat. In those regions in which unstriped muscle is not found normally, as in the abdominal recti muscles, its presence in the laparotomy scar tumors has been attributed to a metaplasia of the surrounding connective tissue cells. In this connection, it is interesting to note that in the metastases to the liver in a case of uterine adenomyoma with carcinomatous degeneration, Dillmann [37] found smooth muscle near large gland tubules.

CASE 5.—*Endometriosis of the Urinary Bladder, Associated with Endometriosis of the Uterus and Fallopian Tube.*

L. C., aged 36, married, gave a normal menstrual history; the last period occurred over two weeks before operation. A right salpingo-oöphorectomy and an appendectomy had been performed a year previously. Her present complaint was pain on the left side and painful and frequent urination, of four months' duration. These symptoms were definitely associated with her periods and were more severe at that time. Cystoscopic examination showed a nodular protuberance at the dome, covered by smooth injected mucosa. At operation, the bladder was found firmly adherent to the top of the uterus. Palpation of the adherent dome of the bladder

68. Ewing, J.: Discussion, Tr. Am. Gynec. Soc., Washington, D. C., May, 1925, Am. J. Obst. & Gynec. **10**:730, 1925.

69. Renisch, H.: Ein Beitrag zu adenomyositis uteri et recti, Ztschr. f. Geburtsh. u. Gynäk. **70**:585, 1912.

70. Nicholson, G. W.: Studies on Tumor Formation, Guy's Hosp. Rep. **76**:188, 1926.

showed a mass, 3 by 4 cm., which was considered malignant. A piece was removed for frozen section diagnosis, which was adenomyoma of the endometrial type. The left fallopian tube was thickened and distorted by dense adhesions, and its fimbriated end was closed. A supravaginal amputation of the uterus, left salpingectomy, and resection of the tumor of the bladder were performed.

On gross examination, the fundus uteri, 7 by 4 by 4 cm., was covered with numerous fibrous tags. From the serosal aspect, a number of bluish cysts projected, averaging 3 mm. in diameter. On section, the myometrium contained numerous small cavities filled with hemorrhagic fluid, and there were also islands of grayish glandular tissue. The fallopian tube, 4 cm. long and 1.5 cm. in diameter, was

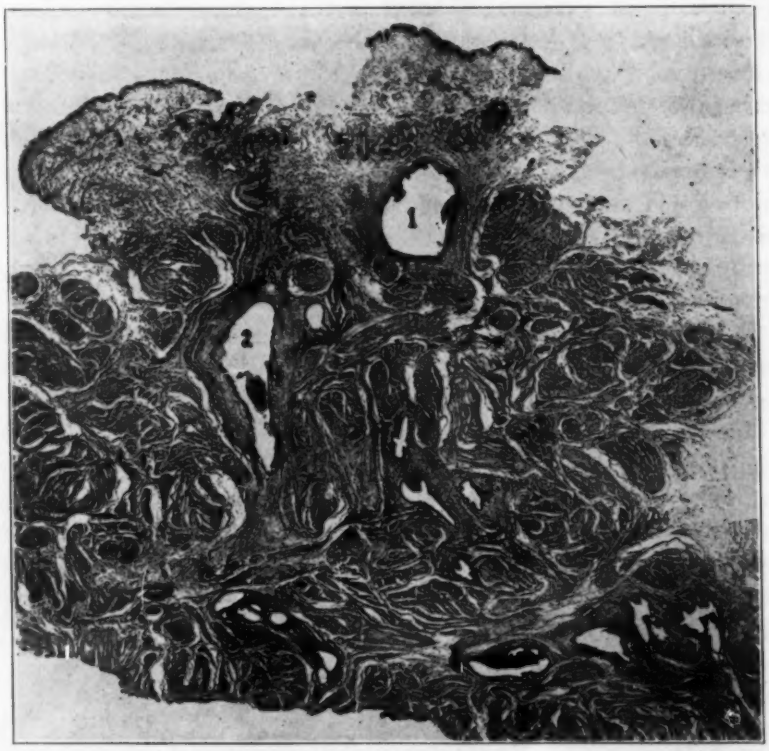

Fig. 15 (case 5).—Endometriosis of the bladder. At the top the bladder is lined by transitional epithelium. Mucosa is edematous and contains many blood vessels. In the muscle wall are many dilated endometrial tubules with more or less cellular stroma. Tubule (1) extends into the mucosa. This and tubule (2) are dilated and still contain some blood. Very low power.

covered with adhesions; its lumen was narrow and the fimbriated end closed. The specimen from the bladder consisted of a firm nodule about 3.5 cm. in diameter. Its cut surface showed dense white fibrous tissue with small blood-filled cysts.

Microscopic examination of the fundus uteri showed a great deal of endometrial tissue scattered through the fibromuscular wall. Characteristic cellular stroma surrounded many of these glandular elements, which nowhere showed any connection with the endometrium. The wall of the fallopian tube and its adhesions con-

tained numerous islands of endometrial tissue. Throughout the wall of the bladder to the very mucosa, there were various-sized gland spaces lined by high cylindric cells, ciliated and nonciliated (fig. 15). These were surrounded by more or less cellular stroma, which was injected and hemorrhagic in places, and here and there in the vicinity of the gland spaces there was blood pigment in endothelial leukocytes.

Comment.—Endometriosis of the wall of the bladder has been observed rarely. We have been able to find only ten cases in the literature. Judd,[71] in 1921, reported a case of adenomyoma presenting as a tumor of the bladder. Here the right cystic ovary and tube and sigmoid were firmly adherent to the tumor mass. As the patient had had two previous pelvic operations in which the uterus, a pelvic tumor and appendix had been removed, one is not sure whether or not this is a case of implantation or direct extension. This is the only case of endometriosis of the bladder among 494 cases of adenomyomas, observed during a course of one and one-half years by Judd and Foulds.[72] In 1925, Keene [73] added two cases of invasion of the wall of the bladder by particles disseminated from perforating ovarian cysts of the Sampson type. The cystoscopic picture was found sufficiently characteristic to permit a preoperative diagnosis of the underlying condition. Brady [74] reported a case of adenomyoma of the vesicovaginal septum with marked urinary symptoms. Heaney's [75] case was associated with adenomyoma of the anterior wall of the uterus and "chocolate cysts" of the ovary. Whitehouse's [76] case occurred in an unmarried woman, aged 22, who had never menstruated. Whitehouse attributes the origin of this growth to a metaplasia of the peritoneal epithelium in spite of the fact that there were dense pelvic adhesions and the tumor occurred in the anterior superior surface of the uterus invading the uterovesical pouch and immovably attached to the bladder. In 1927, there were reported two cases of adenomyosis vesicae by Frommolt,[77] one of which

71. Judd, E. S.: Adenomyoma Presenting as a Tumor of the Bladder, S. Clin. N. Amer. **1**:1271, 1921.

72. Judd, E. S., and Foulds, G. S.: Adenomyomata Involving the Sigmoid, Surg. Gynec. Obst. **37**:648, 1923.

73. Keene, F. E.: Perforating Ovarian Cysts (Sampson's) with Invasion of the Bladder Wall: Report of Two Cases, Am. J. Obst. & Gynec. **10**:619, 1925.

74. Brady, L.: Discussion, Tr. Am. Gynec. Soc., May, 1925; Am. J. Obst. & Gynec. **10**:730, 1925.

75. Heaney, N. S.: Discussion, Tr. Am. Gynec. Soc., May, 1926; Am. J. Obst. & Gynec. **10**:730, 1925.

76. Whitehouse, H. B.: Endometrioma, Invading the Bladder, Removed from a Patient Who Had Never Menstruated, Proc. Roy. Soc. Med. **19**:15, 1925-1926.

77. Frommolt, G.: Zwei Faelle von Adenomyosis der weiblichen Blase, Zentralbl. f. Gynäk. **51**:1159, 1927.

was of intestinal origin, two by Mueller,[78] and one by Morson.[79] As shown in these cases, the endometriosis of the wall of the bladder occurred either by direct extension from an adenomyoma of the anterior uterine wall or by implantation from hemorrhagic cysts of the ovary. A study of table 2 shows that the outstanding symptom was frequent and painful micturition, which occurred during or was aggravated by menstruation. Urgency and urinary tenesmus were also present. Hematuria occurred in only two cases. Two other cases did not show bladder symptoms. The nodular bluish-red appearance of these tumors of the bladder may cause a mistaken diagnosis of malignancy, if endometriosis is not kept in mind.

TABLE 2.—*Endometriosis Involving the Bladder*

No.	Author	Age	Marital	Urinary Symptoms	Effect of Menses	Blood in Urine
1	Judd [72]	34	Single	Frequent and painful micturition; tenesmus	More severe	
2	Keene [73]	40	Married; nullipara	Frequent and painful micturition; urgency	Only during menses	
3	Keene [73]	48	Single	Frequent and burning micturition; urgency	None	Yes
4	Brady [74]	43	Married	Dysuria and polyuria		
5	Heaney [75]	30	.....	Dysmenorrhea and menorrhagia masked bladder symptoms		
6	Whitehouse [76]	22	Single	Severe and increasing abdominal pain at regular monthly intervals	Never menstruated	
7	Frommolt [77]	42	Married; 1 abortion	None		
8	Mueller [78]	30	.....	Painful micturition	During menses	None
9	Mueller [78]	30	Married; nullipara	None		
10	Morson [79]	42	Married	Painless hematuria	.....	Yes
11	Hosoi and Meeker	36	Married	Frequent and painful micturition	More severe	None

CASE 6.—*Particles of Endometrium Lying Free in the Lumen of a Normal Fallopian Tube.*

B. S., aged 41, married, had had three full term pregnancies, one miscarriage and a curettage five years previous to her present illness. Her complaint was sacral backache of three years' duration, frequency and burning urination for one year and leukorrhea for two years. The menses were normal. The last period occurred twenty-one days before operation. She had not had any Rubin test for patency of fallopian tubes at any time. Vaginal examination showed a cystocele, rectocele, lacerated cervix and a prolapse and retrodisplacement of the uterus. Colporrhaphy, perineorrhaphy and right salpingo-oöphorectomy were performed.

78. Mueller, J.: Endometrioide Adenomatose (Adenomyosis) u. Cystadenomatose der Harnblase, Arch. f. klin. Chir. **145**:394, 1927.

79. Morson, A. C.: The Pathology and Treatment of a Vesical Tumor Resembling an Endometrioma, Brit. J. Surg. **15**:264, 1927.

Grossly, the fallopian tube measured 60 mm. in length and from 6 to 10 mm. in diameter. The fimbriated end was patent. Serial sections showed the tube to be normal and covered everywhere by an intact epithelial layer. In the ampulla between the plicae and lying free in the tubal lumen, there were several fragments of definite endometrium and cellular debris (fig. 16). The ovary measured 30 by

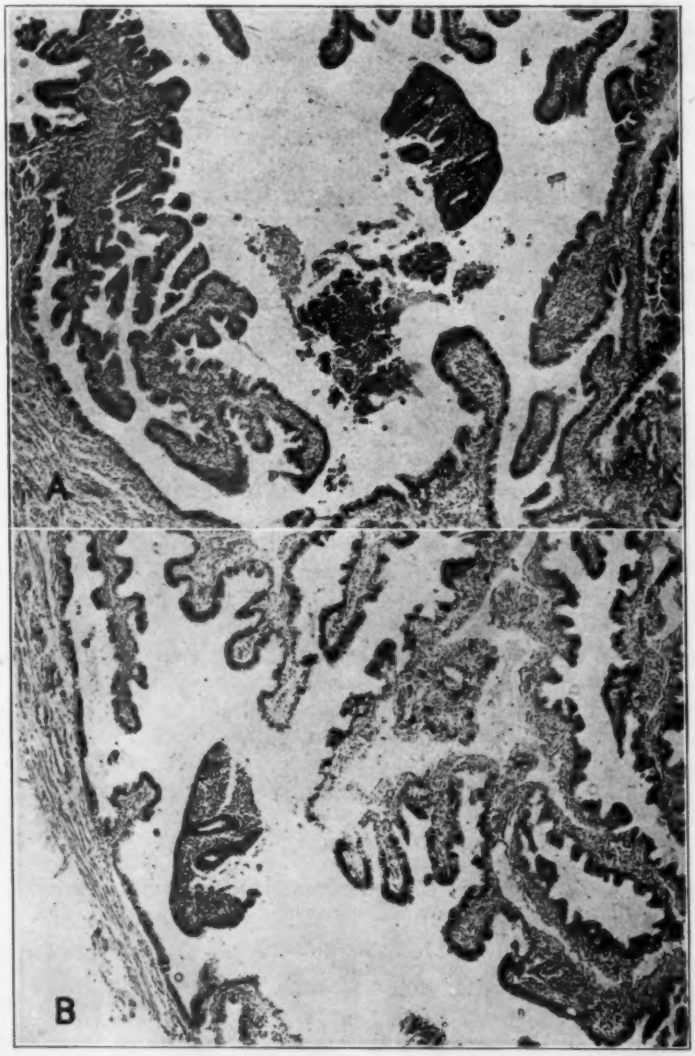

Fig. 16 (case 6).—Fragments of endometrium and cellular debris, lying free in the lumen of a normal fallopian tube. Note that the endometrial particle is not of the menstruating type. This patient was not menstruating at the time of the operation. Medium power.

22 by 15 mm., and contained a ruptured follicle which was hemorrhagic. Serial sections from many blocks of tissue failed to demonstrate any endometriosis in the ovary.

CASE 7.—*Endometrial Particles in the Lumen of the Fallopian Tube Associated with Endometriosis of the Ovary, Tube and Posterior Wall of the Uterus.*

M. C., aged 42, unmarried, had always had normal periods, but for the past five years, there had been menorrhagia. The patient herself noticed a tumor in the lower part of her abdomen, which she was able to palpate. A supravaginal hysterectomy and left salpingo-oöphorectomy were performed.

Grossly, the uterus showed multiple leiomyomas and torn fibrous adhesion bands on the posterior surface. The endometrium was pale, edematous and ecchymotic in some areas. Microscopically, it showed a moderate tortuosity of the gland tubules. In the sections from the posterior wall of the uterus in the area of the adhesion bands, many dilated gland lumina were present. These were lined by a single layer of ciliated and nonciliated cells, varying in form from flat to tall columnar, and surrounded by a more or less moderate amount of cellular stroma. No connection with the endometrium was demonstrable. One gland structure had the appearance of, a pseudoglomerulus of von Recklinghausen. Blood pigment was present, phagocyted by endothelial leukocytes in the stroma or in the gland lumina. The fallopian tube measured 55 mm. in length with a diameter of 7 mm. at the ampulla. The fimbriated end was patent. Microscopically, evidence of an acute or chronic inflammation or its residuum was not present. Lying free in the lumen between the folds were several small groups of exfoliated glandular elements of the uterine type (fig. 17 *A*). Serial sections of the entire tube demonstrated that in the subserosa and subjacent portions of the wall of the midampullary portion there was a myomatous nodule, 3 by 1.5 cm., containing islands of tubules lined by a single layer of tall columnar cells, some of which were ciliated. A narrow zone of cellular stroma surrounded each gland, and blood pigment was present in many of the endothelial leukocytes in one area. The polycystic ovary measured 45 by 30 by 25 mm. Although most of the cysts were filled with clear fluid, one large thin-walled cyst, 27 by 18 mm., contained a tarry fluid. Microscopic examination of this cyst wall showed: (*a*) partly granulation tissue containing numerous endothelial leukocytes with much engulfed blood pigment, and a lining of single-layered, ciliated and nonciliated, cuboidal to tall columnar cells, which had exfoliated in places; and (*b*) partly a cellular stroma, 1 mm. in thickness, containing numerous tortuous gland tubules, which ran directly toward the surface, indistinguishable from a section of the uterine endometrium (fig. 17 *B*). Numerous sections from other portions of the ovary showed minute islands of endometrial-like glands, surrounded by a thin zone of cellular stroma, ecchymotic and containing much blood pigment engulfed by endothelial leukocytes.

Comment.—Foreign cellular particles, benign and malignant, have been infrequently observed in the tubal lumen. Endometrial particles with or without blood, lying free in the tube lumen have been reported by Jaegerroos,[80] Bailey,[81] Lahm,[82] Cordua,[83] Sampson,[84] Novak [85] and

80. Jaegerroos, B. H.: Zur Kenntnis der Veraenderungen der Eileiterschwangerschaft waehrend der Menstruation, Ztschr. f. Geburtsh. u. Gynäk. **72**:28, 1912.

81. Bailey, K. V.: The Etiology, Classification and Life History of Tumors of the Ovary and Other Female Pelvic Organs Containing Aberrant Muellerian Elements with Suggested Nomenclature, J. Obst. & Gynec. Brit. Emp. **31**:539, 1924.

82. Lahm, W.: Schleimhautdurchwanderung durch die Tube, Centrabl. f. allg. Path. **36**:49, 1925.

83. Cordua, R.: Die Moeglichkeit des Transportes intrauterinen Materials in die Tube, Zentralbl. f. Gynäk. **50**:720, 1926.

Meyer.[86] Carcinoma cells were found by Sitzenfrey,[87] Schiller,[88] Sampson[89] and Novak;[85] and sarcoma cells by von Franque.[90] In four of the seven cases reported by Sampson a previous diagnostic curettage had been done, showing that curettage and manipulation of the uterus may force tissue particles from the uterine cavity into the tubes. In one of his cases with extensive carcinoma of the uterus, in one place a piece of cancer was adherent to the tubal mucosa, destroying and replacing it. In all these cases, the primary carcinoma or sarcoma was located in the uterus, demonstrating that these cells have arrived there by the tubal route. Reichel[91] was the first to discuss (1888) the possibility of the tubal route in the dissemination of cancer cells from a focus, primary either in the uterus or in the ovary. That cancer particles also travel downward toward the uterus is seen in one of Sampson's cases of endometrial carcinoma of the ovary in which bits of cancer were found free in the lumen of the tube; the mucosa in the uterus was senile and atrophic.

In our case 6, the particles of endometrium in the lumen could have arrived there only from the uterine cavity, either by mechanical squeezing of the uterus before or during operative maneuvers or by regurgitation through contractions of the uterus. In case 7, in which an associated endometriosis of the ovary, tube and the posterior wall of the uterus was present, the endometrial particles in the lumen could come from the uterine cavity or more likely from any one of the ectopic endometrial foci in the pelvis. In this case, these particles in the tubal lumen were traveling toward the uterus. The regurgitation theory has been championed by Sampson, who has observed menstrual blood dripping from the fimbriated end of the fallopian tube of a

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84. Sampson, J. A.: Peritoneal Endometriosis Due to the Menstrual Dissemination of Endometrial Tissue into the Peritoneal Cavity, Am. J. Obst. & Gynec. **14**: 422, 1927.

85. Novak, E.: The Significance of Uterine Mucosa in the Fallopian Tube, with a Discussion of the Origin of Aberrant Endometrium, Am. J. Obst. & Gynec. **12**:484, 1926.

86. Meyer, R.: Ueber Endometrium in der Tube sowie ueber die hieraus-entstehenden wirklichen und vermeintlichen Folgen, Zentralbl. f. Gynäk. **51**:1482, 1927.

87. Sitzenfrey, A.: Ueber die Verschleppung von Krebskeimen durch die freie Tube, Gynaek. Rundschau **2**:393, 1908.

88. Schiller, W.: Ein Fall von freiliegenden Krebspartikeln in der Tube bei primaeren Karzinom des Corpus uteri, Monatschr. f. Geburtsh. u. Gynäk. **59**:307, 1922.

89. Sampson (footnote 1). Surg. Gynec. Obst. **38**:287, 1924.

90. Von Franque, O.: Ueber Sarcoma uteri, München. med. Wchnschr. **45**: 1301, 1898; Ztschr. f. Geburtsh. u. Gynäk. **40**:183, 1899.

91. Reichel, P.: Ueber das gleichzeitige Vorkommen vono Carcinom des Uteruskoerpers und des Eierstockes, Ztschr. f. Geburtsh. u. Gynäk. **15**:354, 1888.

Fig. 17 (case 7).—In *A*, small clumps of exfoliated columnar cells are present, lying free in the tubal lumen. In *B*, is shown an endometriosis of the ovary. The section was taken from the wall of a hemorrhagic cyst (Sampson type). Note the definite lining of characteristic cellular stroma and early tortuous gland tubules, all running directly to the surface. It is indistinguishable from the uterine mucosa. Medium power.

menstruating patient during the course of a gynecologic operation. In 1888, Minard [92] observed dark menstrual nonfibrous flow passing out, drop by drop, from the tubes of a menstruating woman who had an inverted uterus. When the tubes were pressed on, there would be a stream for an instant. In thirteen operations on menstruating women, Novak [85] never observed blood dripping from the tubal orifices. Czyzewicz [93] studied six cases, two during menstruation and four during the intermenstrual period, and found that during the catamenia typical blood elements could be seen in the lumen of the healthy tube which was not the case during the intermenstrual interval. In Schmid's [94] patient, who had marked menstrual bleeding causing severe anemia, a small blood clot in the ampulla of a tube and a small amount of blood in the peritoneal cavity were observed. The uterus and tubes were normal. Schmid concluded that a back flow of menstrual blood could occur not only in myomas, retroflexion and other conditions, causing more or less obstruction of the lower uterine canal, but also in the absence of pathologic conditions through uterine contractions. That uterine menstrual blood may pass into the tubes is shown in those cases of Bland,[95] Thorn,[96] Behrend,[97] Mueller,[98] Ballin [99] and others, in which a bloody discharge occurred periodically from a fistulous abdominal opening which communicated with the uterine cavity by way of the tubal lumen. Davis and Cron [100] reported a case of pelvic endometriosis, probably due to intra-uterine inflation with gas.

According to Novak,[85] the large pieces of endometrial tissue, as have been observed in the tubal lumen, cannot possibly pass through the narrow interstitial portion of the fallopian tube, which has normally a diameter of from 0.5 to 1.0 mm. at the uterine orifice. Zorn [101] investi-

92. Minard, E. J. C.: Does the Menstrual Flow Originate in the Tubes? The Act of Menstruation Viewed from an Inverted Uterus, Tr. New York M. A. **5**: 185, 1888.

93. Czyzewicz, A.: Zur Tubenmenstruation, Arch. f. Gynäk. **85**:197, 1908.

94. Schmid, H. H.: Blutaustritt aus der Tube waehrend der Menstruation, Zentralbl. f. Gynäk. **49**:44, 1925.

95. Bland, P. B.: Tubal Menstruation: A Case History, Therap. Gaz. **19**:731, 1903.

96. Thorn, W.: Zur Frage der Tubenmenstruation, Zentralbl. f. Gynäk. **28**: 971, 1904.

97. Behrend, M.: Beitrag zur Frage des Blutaustritts aus der Tube waehrend der Menstruation, Zentralbl. f. Gynäk. **49**:1007, 1925.

98. Mueller, P. P.: Beitrag zur Frage des Blutaustritts aus der Tube waehrend der Menstruation, Zentrabl. f. Gynäk. **49**:1977, 1925.

99. Ballin, M.: Menstrual Fistula of Postoperative and Endometrial Origin, Surg. Gynec. Obst. **46**:525, 1928.

100. Davis, C. H., and Cron, R. S.: A Contribution to the Study of Endometriosis, Am. J. Obst. & Gynec. **2**:526, 1926.

101. Zorn, W.: Ueber die Lumenweite der Pars interstitialis des Eileiters beim Menschen, Zentralbl. f. Gynäk. **50**:3064, 1926.

gated eighty-four tubes, using only fresh operative material. He found that during the sexually mature age, the diameter of the pars interstitialis was from 0.7 to 1.0 mm.; during gravidity, from 0.8 to 1.0 mm., and during the preclimacterium and menopause, from 0.7 to 0.9 mm. In the fifty cases examined by Rubin [102] by means of injections of iodized oil 40 per cent and the x-ray, the tube emerged from the uterine cavity in a straight course; the intramural portion of the tube had the power of dilating and contracting; and in the presence of a foreign body, like iodized oil, reversed peristalsis was not uncommon. Owing to this dilatation and contraction of the tube, and owing to the compressibility or molding of the endometrial tissues, particles larger than the largest tubal diameter of 1 mm. may be forced through this intramural portion by powerful uterine contractions or by mechanical squeezing during gynecologic maneuvers.

Can the blood originate from a true tubal menstruation? That the normal healthy tubal mucosa menstruates in the sense of hyperplasia, hemorrhage and exfoliation is denied by most investigators. In those cases in which free blood was found in the tubal lumen, evidence of diapedesis or of hemorrhage into the tubal mucosa could not be demonstrated anywhere. The studies of Holzbach,[103] Czyzewicz, Jaegerroos and Snyder [104] show that cyclic changes do occur in the normal tubal mucosa, but no menstrual phenomena can be observed such as occur in the endometrium. As the blood can come from the uterine cavity, Lahm [105] pointed out that a true tubal menstruation can only be determined through histologic examination of the fallopian tube. Hoehne,[106] Schwartz and Crossen,[107] Schridde and Schoenholz,[108] Schindler,[109]

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102. Rubin, I. C.: Observations on the Intramural and Isthmic Portion of the Fallopian Tubes with Special Reference to the So-Called "Isthmospasm," Surg. Gynec. Obst. **46**:87, 1928.

103. Holzbach, E.: Vergleichend-anatomische Untersuchungen ueber die Tubenbrunst und die Tubenmenstruation, Ztschr. f. Geburtsh. u. Gynäk. **61**:565, 1907-1908.

104. Snyder, F. F.: Changes in the Fallopian Tube During the Ovulation Cycle and Early Pregnancy, Bull. Johns Hopkins Hosp. **34**:121, 1923; Changes in the Human Oviduct During the Menstrual Cycle and Pregnancy, ibid. **35**:141, 1924.

105. Lahm, W.: Zur Frage der Tubenmenstruation auf dem Boden einer endometoiden Fehlbildung ihrer Schleimhaut, Arch. f. Gynäk. **130**:152, 1927.

106. Hoehne: Untersuchungen der Pars interstitialis der Tube, Ber. Nordwest deutsche Ges. f. Gynaek., Oct. 27, 1923; Zentralbl. f. Gynäk. **48**:233, 1924.

107. Schwartz, O. H., and Crossen, R.: Endometrial Tissue in the Ovary, Am. J. Obst. & Gynec. **7**:505, 1924.

108. Schridde, H., and Schoenholz, L.: Epitheliofibrose und Epitheliomyose der Eileiter, Frankfurt Ztschr. f. Path. **30**:338, 1924.

109. Schindler, B.: Uterusschleimhaut in der Tube, Zentralbl. f. Gynäk. **49**: 582, 1925.

Meyer [110] and Lahm described a partial to complete replacement of the tubal mucosa by typical endometrium, which reacted to the ovarian hormone at the menstrual period. In these cases of true tubal menstruation, particles of cast-off endometrium and menstrual blood can travel either toward the uterus or into the peritoneal cavity to become implanted there. When the fimbriated end is occluded, a hematosalpinx may form, due to the successive pent-up menstrual flow, as in the cases of Schindler and Meyer. Thus, one sees that endometrial particles and blood in the tubal lumen may come from the uterus, from the tube itself, or from an ectopic endometrial focus in the pelvic cavity.

CONCLUSIONS

1. Seven unusual cases of endometriosis are reported: (*a*) in an inguinal lymph node, associated with endometroisis of the groin and endometrial carcinoma of the transverse colon; (*b*) of the veriform appendix, associated with endometriosis of the fallopian tube; (*c*) of the veriform appendix with no other demonstrable pelvic pathologic process; (*d*) in an appendectomy scar, twenty-six and one-half years after the appendectomy; (*e*) of the urinary bladder, associated with endometriosis of the uterus and fallopian tube; (f) particles of endometrium lying free in the lumen of a normal fallopian tube, without associated pelvic pathologic process, and (*g*) endometrial particles in the lumen of the fallopian tube, associated with endometriosis of the ovary, tube and posterior wall of the uterus.

2. The various theories of pathogenesis and the symptomatology, discussed in connection with the cases, indicate that no one theory can be used to explain all the observations.

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110. Meyer, R.: Ueber adenomatose Schleimhautwucherungen in der Uterus- und Tubenwand und ihre pathologisch-anatomische Bedeutung, Virchows Arch. f. path. Anat. **172**:394, 1903.

THE TREATMENT OF ABSCESS OF THE BRAIN*

C. C. COLEMAN, M.D.
RICHMOND, VA.

The pathologic changes in localized intracranial suppuration vary widely and, for the sake of accuracy, the type of abscess should be specified before advantages for any particular method of surgical treatment are claimed. Multiple pyemic abscesses are nearly always fatal and do not present any aspects of great surgical interest (figs. 1 and 2). Abscesses due to gunshot wounds, if solitary, have a better prognosis, but even these have essential causes for a high mortality (fig. 3). Infection by highly virulent organisms may produce a rapid necrosis which the tissues of the brain are unable to encapsulate and resist. Treatment for this type of abscess is generally unsuccessful.

The material in this paper is restricted entirely to the encapsulated, subcortical abscess which usually arises from infection of the accessory sinuses. These abscesses, if successfully resisted by the brain tissues, pass through a stage of encephalitis which is followed by the formation of a capsule. With formation of the capsule, systemic signs of infection are likely to become quiescent, but it is believed that it is exceptional for such cases to be free from cerebral symptoms, and therefore, there is a well-founded objection to characterizing the stage of encapsulation as a "latent" or "quiescent" stage of the abscess. It is difficult to recognize the first stage of abscess, and this is not of great importance surgically, because operation should not be done until after the formation of a capsule. The results of operation are just as likely to be good in old abscesses as in the early stages of encapsulation, provided rupture has not taken place.

A review of the literature indicates a great improvement in the mortality statistics of encapsulated abscess of the brain, and it is probable that McEwen's [1] statement in 1893 that it is one of the most satisfactory of intracranial lesions is likely to be verified in future experience.

There are now four recognized operative procedures for the relief of abscess of the brain: (1) drainage of the abscess through a small trephine opening with a short incision in the dura, and immediate or delayed insertion of the eye-end of a soft rubber catheter into the abscess

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* From the Department of Neurological Surgery, Medical College of Virginia.

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1. McEwen, William: Pyogenic Infective Diseases of the Brain and Spinal Cord, New York, the Macmillan Company, 1893.

cavity; (2) the osteoplastic flap exposure of the brain with drainage of the abscess, as advocated by Eagleton;[2] (3) single or repeated tapping of the abscess without drainage of any kind, as recommended by Dandy,[3] who expressed the belief that in encapsulated abscess the brain needs only a relief of tension to enable it to overcome the infection; (4) unroofing the abscess; that is, cutting away the cortex which overlies the abscess, as proposed by King,[4] in 1924. King believed that by his method, the abscess is averted by the coincident intracranial pressure. Drainage of any kind is therefore unnecessary in King's procedure.

In the enthusiasm for certain types of drain and ingenious devices to promote escape of pus from an abscess of the brain, important pathologic

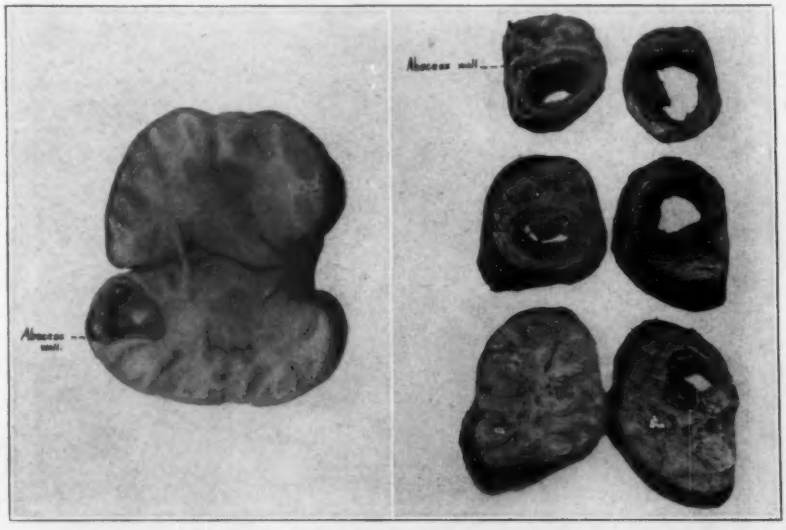

Fig. 1.—Multiple abscesses, seven in number, arising from an undiscovered source. These abscesses were bilaterally located in the temporal, occipital and parietal lobes; they were streptococcic in type and were well encapsulated, as shown in the sections of the brain. Autopsy was limited to the head.

factors in the results of treatment seem to have been overlooked. It will be readily appreciated from the wide difference of opinion as to the preferable technic of eradicating the abscess that this phase of the

2. Eagleton, W. P.: Brain Abscess, New York, the Macmillan Company, 1922.

3. Dandy, Walter E.: Treatment of Chronic Abscess of the Brain by Tapping, J. A. M. A. **87**:1477 (Oct. 30) 1926.

4. King, J. E. J.: The Treatment of Brain Abscess by Unroofing and Temporary Herniation of Abscess Cavity with the Avoidance of Usual Drainage Methods; with Notes on the Management of Hernia Cerebri in General, Surg. Gynec. Obst. **39**:554, 1924.

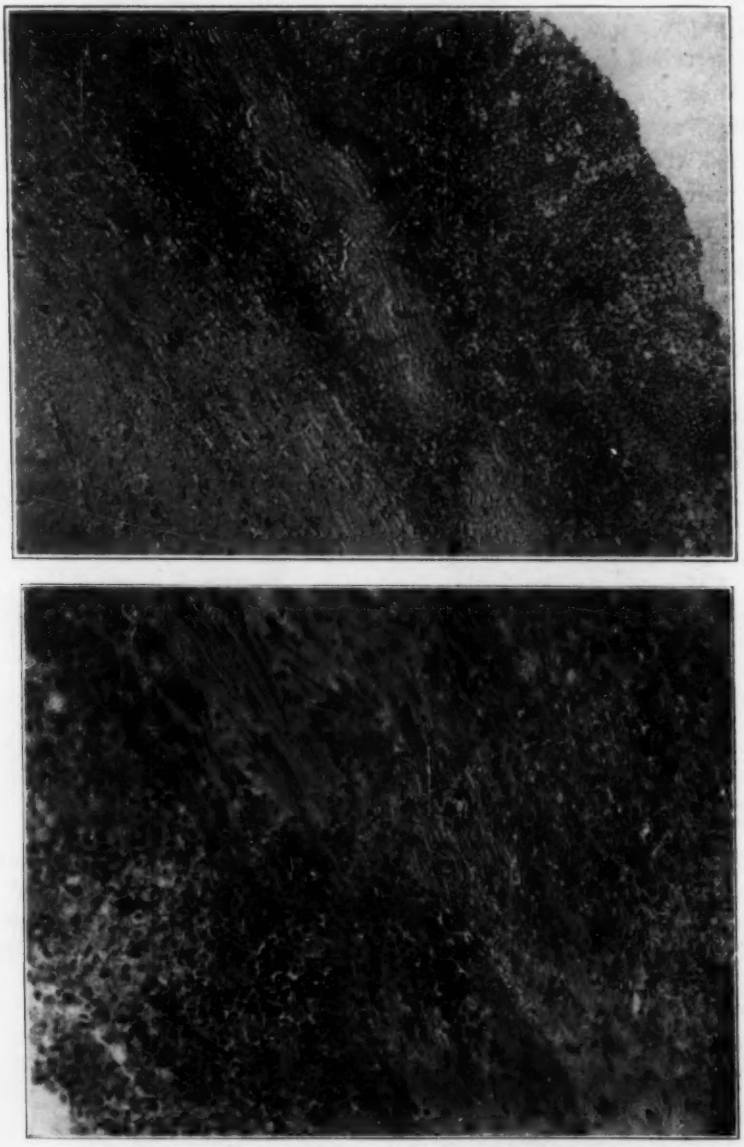

Fig. 2.—Sections from the abscess wall of a metastatic abscess shown in figure 1. It is composed of layers of firm chronic inflammatory tissue about 1 mm. thick, well vascularized and infiltrated with lymphocytes. The contents of the abscess were partially autolyzed pus and cellular débris. The brain tissue immediately adjacent is compressed and somewhat degenerated.

treatment is still unsatisfactory and that there are inherent risks attending drainage of any kind. In the series of cases considered in this paper, simple drainage with the eye-end of a soft rubber catheter has been used. The operation is nearly always done under local anesthesia, and the drainage tube is inserted into the abscess cavity when the pus is first discovered, if great difficulty is not encountered in the insertion of the tube.

A partial review of the literature does not show any important advance in the treatment for abscess of the brain since McEwen published his monograph in 1893, with the exception of the use of local

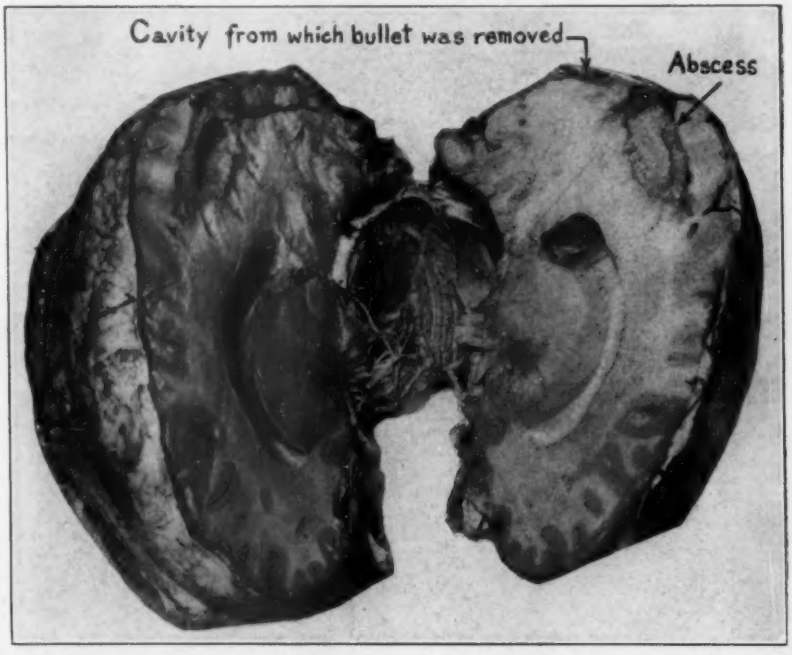

Fig. 3.—Specimen from patient who had a right hemiplegia with a disturbance in speech which had existed since he received a gunshot wound two months before coming under observation. There had been no progression of symptoms, and the abscess was not suspected or found when the bullet was removed under local anesthesia. The bullet lay just above the left lateral sinus and was well encysted. Apparently the cavity containing the bullet did not communicate with the abscess, which lay further forward. In the removal of the bullet, the wall of the abscess was evidently disturbed, allowing the escape of pus and causing death from meningitis in thirty-six hours. A culture of the bullet was negative for organisms, but the spinal fluid later showed a streptococcic meningitis. The abscess was located in the left occipital and parietal lobes.

anesthesia, which is now in more or less general use. At that time, McEwen reported nineteen cases of cerebral and cerebellar abscess, with

eighteen recoveries. As far as is known, such a record has not been equaled by any of his contemporaries or followers. McEwen laid great stress on cleansing the abscess cavity, and he advocated closure of the wound without drainage in some cases of encapsulated abscess. He never used drainage in the acute cases. I cannot see any particular reason for the employment of an osteoplastic flap, which of itself may be quite a formidable procedure in patients suffering from severe compression. Eagleton expressed the belief that an osteoplastic flap permits a wider exploration of the cortex, and that collections of pus in the

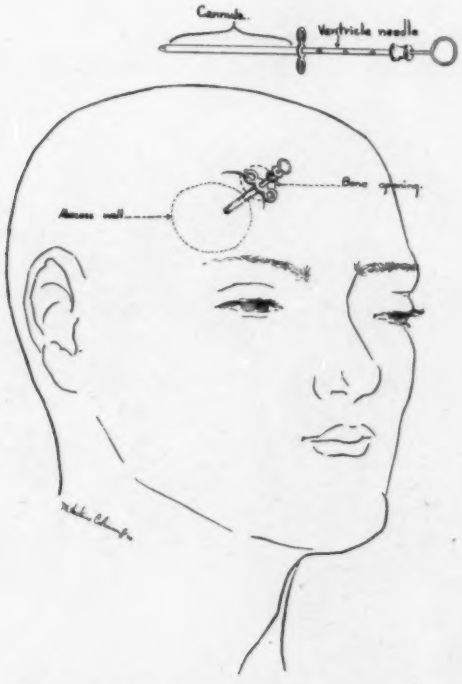

Fig. 4.—Suitable location for puncture of a frontal abscess. Above is shown the graduated ventricle needle with a cannula. The cannula may be left in the abscess cavity if it is deeply situated, to prevent "losing" the abscess.

pia-arachnoid, which otherwise might be overlooked, are discovered. I have not found collections of pus in the pia-arachnoid, and when a patient has signs of general intracranial pressure due to abscess, it is probable that the abscess is subcortical. It is much simpler and safer to make several explorations through a trephine opening in various areas of the brain in the search for abscess than it is to make an osteoplastic flap. Occasionally, an osteoplastic flap may be done because of the impossibility of differentiating between an abscess and a tumor. An osteoplastic flap was made in two such cases, as it was thought that a glioma was present.

The method which has been practiced for several years, with encouraging results, is as follows:

After the abscess is localized by neurologic study of the patient, a small drill opening is made in the bone and the dura incised for a distance of about 1 cm. In the cases in which this was done, there was considerable pressure, and the dural incision was promptly plugged by the cortex. A ventricle needle over which a cannula is fitted is passed into the brain, and the abscess wall is easily recognized in most cases by the resistance to the advancing needle. Sometimes considerable force is necessary to push the ventricle needle through the capsule into the abscess. A small amount of pus is allowed to escape through the

Fig. 5 (case 9).—Large temporal fungus (photograph) following subtemporal decompression for tumor. The patient had three abscesses, indicated in the drawing of the brain specimens as 1, 2 and 3. The location of the small burr opening made for drainage of the abscess in the right frontal lobe is indicated by *A*, which shows a small flap of the dura attached to the brain around the puncture. Abscess 3 was evacuated, but 1 and 2 were not discovered until autopsy. Death occurred in six weeks from septic encephalitis.

needle. If the abscess is at a great distance from the surface (as much as 5 or 6 cm.), the abscess is sometimes sought by a second approach, the first needle being left in position until the second reaches the abscess through a shorter route. If it is decided that drainage is to be made through the first trephine opening and the abscess is at a great depth, particularly if the capsule is

dense, the cannula is pushed into the abscess cavity, the needle withdrawn and the cannula allowed to remain for drainage (fig. 4). On several occasions, difficulty was encountered in inserting a catheter into the abscess cavity after withdrawal of the ventricle needle. This formerly caused great concern, for at this stage processes leading to the death of the patient are most likely to be initiated. Precipitate efforts at drainage with large, traumatizing drainage tubes are likely to push the capsule ahead of the tube, allowing the pus to seep through the opening made by the needle into the brain tissues outside of the abscess wall. The resistance of these tissues, suddenly invaded by pyogenic organisms, is overcome and a septic encephalitis is initiated. I do not agree with Dowman [5] that patients who die following operations for abscess of the brain generally die of meningitis, but that they have a septic encephalitis provoked by the surgeon's well-intended efforts, and that while meningitis may be a terminal event, the primary cause is encephalitis.

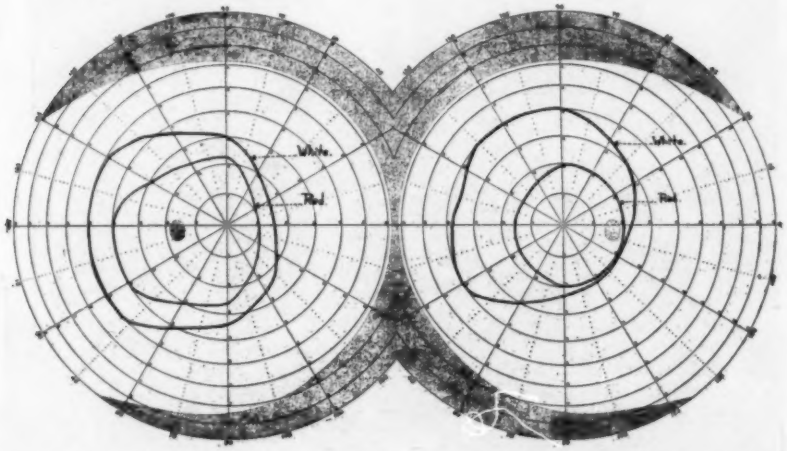

Fig. 6. (case 6).—Extensive defect in right field due to abscess of left temporal lobe.

If difficulty is encountered now in inserting the drainage tube into the abscess cavity, one should not hesitate to close the wound entirely without drainage of any kind and wait for five or six days before attempting to insert the soft rubber catheter. In two recent cases this was done, and evidently softening of the capsule had taken place at the point of entrance of the ventricle needle, because the same type of drainage tube went into the abscess cavity without difficulty, through the same track, at the second attempt. I prefer to use a small drainage tube when it can be expertly and promptly placed into the abscess, but if there is much fumbling with the drainage tube and great difficulty is experienced in inserting it into the cavity, one should adopt the method of simple tapping without drainage, as advocated by Dandy. The most important feature in the treatment for abscess of the brain is the drainage of the abscess with the

5. Dowman, Charles E.: The Treatment of Brain Abscess by the Induction of Protective Adhesions Between the Brain Cortex and the Dura Before the Establishment of Drainage, Arch. Surg. **6**:747 (May) 1923.

least traumatizing form of drainage material and with the least difficulty. It is highly probable that a number of encapsulated abscesses may be eradicated by repeated tapping, and one must be impressed by the fact in some cases that after the primary evacuation of pus, there is slight subsequent drainage, which may be largely due to reaction of the tissues to the presence of the drainage tube, and probably little or none comes from the abscess cavity. If, however, a small catheter is passed into the abscess cavity without difficulty, it should provide a factor of safety—not a part of simple tapping. In the cases in which McEwen closed the wound tightly, he emphasized the importance of removing completely all débris and pus from the abscess. But even such a radical procedure could not have removed the limiting pyogenic membrane, and after all it is probable that the important factors of success in the results of his treatment are identical with those of simple tapping.

In 1924, King described three patients successfully treated by cutting away the cortex and allowing the abscess to extrude. McEwen recommended this technic in certain cases, but he did not give the same explanation of its advantages as King did. It does not appear that this method is necessary in the majority of cases, nor that it would be applicable to deep-seated abscesses. The object of King's method is to allow the abscess wall to be turned inside out by the intracranial pressure. This results in a fungus, which in itself is not a serious condition, provided it is not due to a progressive septic encephalitis. The method of King would seem to be more appropriate in extensive abscesses near the cortex, with necrosis of brain tissue and imperfect or no capsule formation. No form of drainage is satisfactory in such cases.

Fig. 7 (case 3).—Right temporal abscess. The origin of the infection was unknown. No focal symptoms but marked signs of general intracranial pressure were present. Localization was made by ventriculograms which showed a collapsed right ventricle. Complete recovery occurred. The arrow indicates the location of the drainage tube.

Fig. 8 (case 7).—*A*, fungus following osteoplastic flap operation for large right frontal abscess. In view of the duration of cerebral symptoms for about fourteen months, the patient was thought to have had a right frontal tumor. In this case there was a high grade of choked disk in the left eye. The right eye had been accidentally destroyed several years before. *B*, patient one year after operation. He appeared to be symptom-free and was busily engaged in the practice of law. The deforming scar was the result of long continued osteomyelitis and infection of the scalp following the flap operation.

Fig. 9 (case 5).—*A*, location of the small catheter used for drainage of the abscesses of the left temporal lobe. The patient had an old mastoid infection. *B*, appearance of patient about one year after operation. After eradication of the abscesses, a radical mastoid operation was done, with complete recovery.

Much success in the treatment for abscess of the brain has been attributed by Cahill [6] of Boston to the Mosher cone-shaped, wire-mesh drain. Cahill advocated exposure of the dura and packing its outer surface with mercurochrome gauze for from forty-eight to seventy-two hours before the drain is inserted. The object of this packing is to produce adhesions between the dura and the underlying arachnoid. It is hardly probable that packing of an unopened dura would produce such adhesions, nor do I believe it necessary to have adhesions between the dura and the underlying membranes. From the description and photographs of the conical, wire-mesh drain, it would seem that it is unnecessary in the evacuation of an abscess and that it would increase the trauma

Fig. 10 (case 4).—Appearance of patient four years after operation for left temporal abscess. The primary infections in the history were parotitis and typhoid fever about two months before intracranial symptoms appeared.

to the surrounding brain tissues. However, I have not had any experience with this method. Cahill's results were exceptionally good.

Especially favorable types of encapsulated abscess arise from mastoid infection and are more or less superficial and are often evacuated through the mastoid wound. The good results of treatment in these cases is due in a large measure to the completeness of primary evacuation, the absence of extensive trauma and the effects of gravity in promoting the continued escape of pus.[7] All kinds of drainage have been used in these abscesses,

6. Cahill, Harry P.: Twelve Cases of Cerebral and Cerebellar Abscesses Drained by the Mosher Wire Gauze Cone, Tr. Am. Otol. Soc. **17**:42, 1925.

7. Coleman, C. C.: Some Observations on the Drainage of Subcortical Brain Abscess, Arch. Surg. **10**:212 (Jan.) 1925.

with little adherence to established neurosurgical technic, and yet a high percentage of recovery results. The treatment for this type of abscess, which is usually close to the surface of the brain and often has a so-called stalk, does not offer a critical test of a technic for the cure of abscess in general. It is in the deep-seated abscesses that an error of technic is usually followed by a fatality from septic encephalitis, and if such cases can be eradicated satisfactorily, there will be little difficulty in the treatment of the more superficial ones. The importance of the operative treatment itself should not cause one to lose sight of the after-treatment in these cases. Time and again I have been greatly disturbed

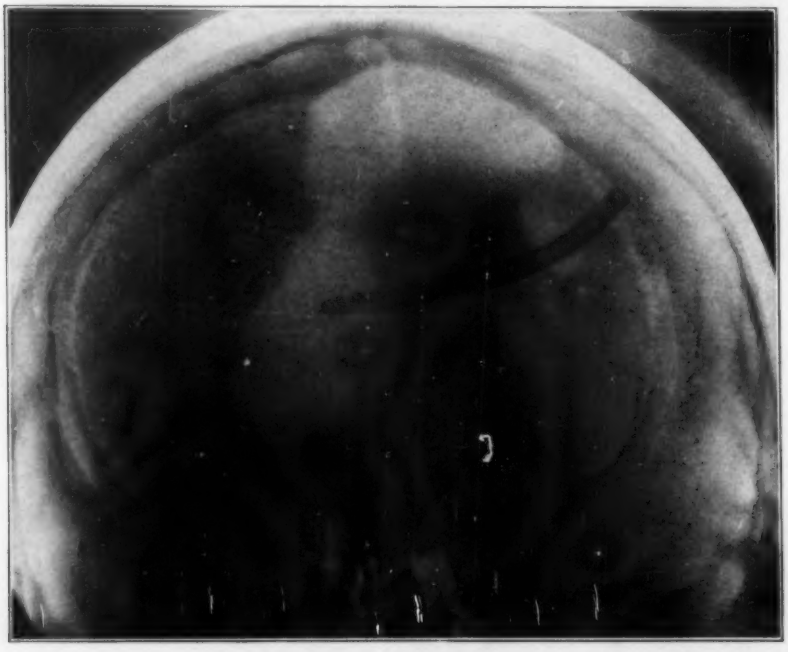

Fig. 11 (case 11).—Destruction of frontal and parietal bones. The drainage tube appears to go beyond the median line. It is this type of abscess which offers a critical test of any form of treatment, and would not seem suited to the method of King nor the gauze-mesh drain of Mosher. When abscesses at this depth are drained, particularly if much trauma is produced to the surrounding healthy brain tissue, an extensive septic encephalitis is likely to result. The drainage tube shown in the roentgenogram was not placed in the abscess cavity until one week after the tapping of the abscess with a ventricle needle.

by the course of the patient's recovery, and I believe that the good results finally obtained in these cases were due to the refusal to make any further attempts at drainage. The after-treatment in these cases is of the greatest importance. Once the drainage tube is in position, it is highly desirable that it should be left until there is no further need for

it. In some cases, after more or less complete evacuation of the abscess with obliteration of the cavity by intracranial pressure, the drainage tube is extruded in a few days. To attempt to replace it with force seems to be unwise.

From experience in a series of encapsulated abscesses and an analysis of the experiences of others, the following conclusions have been tentatively reached: 1. There are a number of types of localized intracranial suppuration, and treatment suggested for one may be entirely unsatisfactory in another of these types. An encapsulated abscess offers a good prognosis if accurately localized and properly treated. Under these circumstances, I believe that recovery should be the rule in this type of abscess. 2. The cause of death from abscesses operated on is nearly

Fig. 12 (case 10).—Abscess of left frontal lobe drained through a small opening in the dura in the upper part of an incision made for an osteoplastic flap. Photograph of patient one year after operation shows a discharging sinus at the lower angle of the incision, due to osteomyelitis. The patient had two general convulsions about six months after leaving the hospital, but for the past four years he has been free from symptoms and actively engaged in business.

always septic encephalitis brought on by efforts at drainage. There appears to be little danger of a fatality after partial collapse of the abscess, provided pus does not escape from the abscess into the surrounding brain tissue. 3. I prefer to drain encapsulated abscess but I believe that this drainage should be done through a trephine opening made under local anesthesia and that the type of drainage best suited, in my experience, is a small, soft rubber catheter. It is important to approach the abscess at the point nearest to the cortex, but such a point cannot always

be determined. I believe that simple tapping of an encapsulated abscess is to be preferred to drainage, if drainage is accomplished under great difficulty or when many attempts are necessary to insert the drainage tube into the abscess cavity.

In the fourteen cases of chronic abscess on which the observations in this paper are based, there were two deaths. A brief summary of the fatal cases is of some interest.

REPORT OF CASES

CASE 9.—A man, aged 25, was admitted to the Memorial Hospital with a large right temporal fungus (fig. 5). Because of a diagnosis of high intracranial pressure from tumor of the brain, a right subtemporal decompression

Fig. 13 (case 11).—Deep left frontal abscess reached through a trephine opening at a depth of 5.5 cm. The abscess followed infection of both frontal sinuses with extensive destruction of the frontal and parietal bone along the midline. The photograph shows the scarring of the scalp following operations for osteomyelitis. The end of the small drainage tube with a safety-pin attached is seen about the hair-line in the left frontal region. The dark spot in the midline of the frontal region is a discharging sinus. The patient, a boy, aged 14, was desperately ill from a metastatic infection, which practically destroyed his right elbow, several weeks before admission to the hospital for the treatment of intracranial symptoms. At the time of admission, he showed marked intracranial pressure with bilateral choked disk but no localizing signs other than a slight drooping of the right upper lip on emotional expression. In the diagnosis of the side involved preference was given to the left side, notwithstanding the fact that both sinuses had been operated on and there was more destruction of bone on the right. This photograph was made on May 24, 1927, one month after the operation for abscess. Complete recovery occurred without residual paralyses. A plastic operation has been advised for the scars in the frontal region.

had been performed in another city. This operation was followed by a breaking down of the wound and protrusion of brain tissue. The patient gave a history of chronic infection of the right frontal sinus. At the time he was admitted to my service, he had a violent headache and a choked disk of 5 diopters with retinal hemorrhages and was stuporous. Fever, leukocytosis and other signs of encephalitis were present. The history indicated a right frontal abscess. An exploration of the right frontal lobe was made through a trephine opening and a thin-walled abscess discovered at a depth of 5 cm. About 15 cc. of pus contained staphylococci was evacuated. Some temporary relief followed drainage of the abscess, but the encephalitis continued. The patient died about six weeks

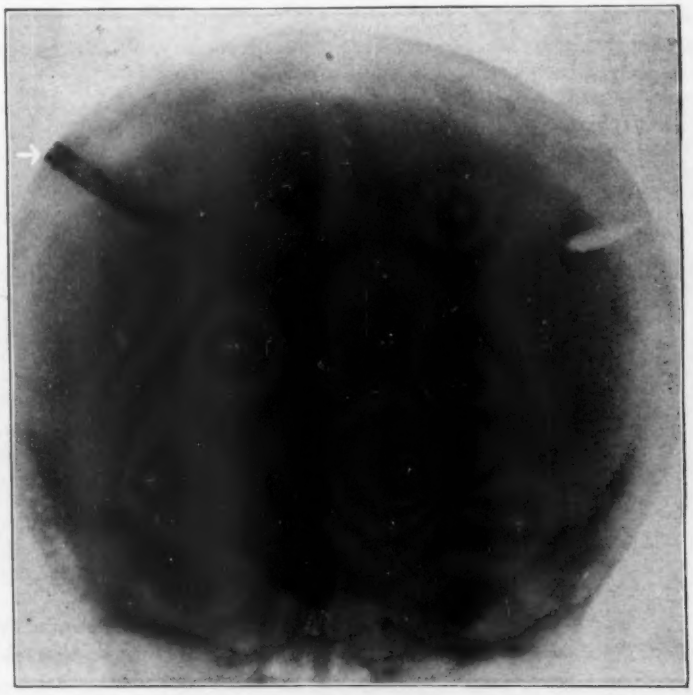

Fig. 14 (case 12).—The arrow points to a drainage tube in the right parietal lobe in a child about 19 months of age. The abscess resulted from the perforation of the skull by a nail four weeks before admission to the hospital. It should be noted that the tube appears to pass slightly beyond the median line.

after admission. Autopsy showed three abscesses, one of which had been drained. There was an extensive encephalitis with necrosis of the brain tissues in the temporal and frontal lobes.

Case 6.—A man, aged 43, was admitted to the hospital with meningitis. The history showed that he had aphasia for seven weeks, and at times was unable to call the names of his children. Severe frontal headache was constant and infrequently was associated with nausea and vomiting. Ataxia of the right arm and a fine tremor of the extended fingers had been noted. During a more coopera-

tive period, a right homonymous hemianopia was demonstrated, but papilledema was not present (fig. 6). Spinal punctures had been done, and these showed considerable pressure, with an excess of globulin in the fluid. Leukocytosis had not been present until the onset of meningitis. The pulse rate had ranged from 50 to 70. Marked weakness of the right side with dilatation of the left pupil further accentuated a lesion in the left hemisphere. The tonsils were infected, but there was no evidence of disease of the mastoid or other sinuses. The patient was referred to my associate, Dr. Lyerly, after the onset of a purulent meningitis, as determined by lumbar puncture before the operation. A ruptured abscess of the left temporal lobe was discovered and drained under local anesthesia. Death followed thirty hours later. This patient was the only one in the series in whom definite effects on the visual fields had been determined before operation. In the majority of the cases, the abscess was so located or else the patient so uncooperative that satisfactory perimetric examinations could not be done. All of the patients with the exception of this one had choked disk.

Fig. 15 (case 13).—*A*, abscess in the right parietal lobe from compound depressed fracture of skull with laceration of longitudinal sinus. The photograph shows the large brain fungus. Incision is shown for right subtemporal decompression to reduce pressure and thus diminish the size of the fungus. The fungus was protected by a doughnut pad, and repeated spinal punctures were followed by reduction in size of the extruded brain tissue. *B*, appearance of child one year later showing recovery from abscess. The destruction of brain tissue in the right hemisphere produced a spastic paralysis of the left arm and leg, which was permanent.

Case 3.—It was necessary to inject air in case 3 (fig. 7) to determine the side involved. A girl, aged 9 years, did not present a history of any previous infection except a small pustule on the knee three months before admission to the hospital. At this time she showed signs of high intracranial pressure. Practically no evidence of involvement of the pyramidal tract was present. The child was stuporous and uncooperative. A ventriculogram showed the left ventricle to be dilated, with a shifting to the left, and no air entered the

right. There was one feature of particular interest in this case: when the temporal bone was exposed, pus was found to be exuding through a pinpoint opening in the field of operation. In no other case of the series was a spontaneous evacuation of pus through the bone discovered.

CASES 5 AND 7.—In these two cases, an osteoplastic flap was made. Three operations were necessary in case 7 before the abscess was finally discovered. About two months intervened between the first and third operations, and the pus was finally evacuated through the puncture made at the second operation. A troublesome osteomyelitis with ultimate recovery followed the osteoplastic operation. The largest abscess of the series was in case 7. The patient gave a history of intracranial disturbance for at least fifteen months; during most of this time he was actively engaged in the practice of law. The previous infection of the frontal sinus had not been given due consideration in the analysis of the symptoms, and the patient was thought to have a glioma of the

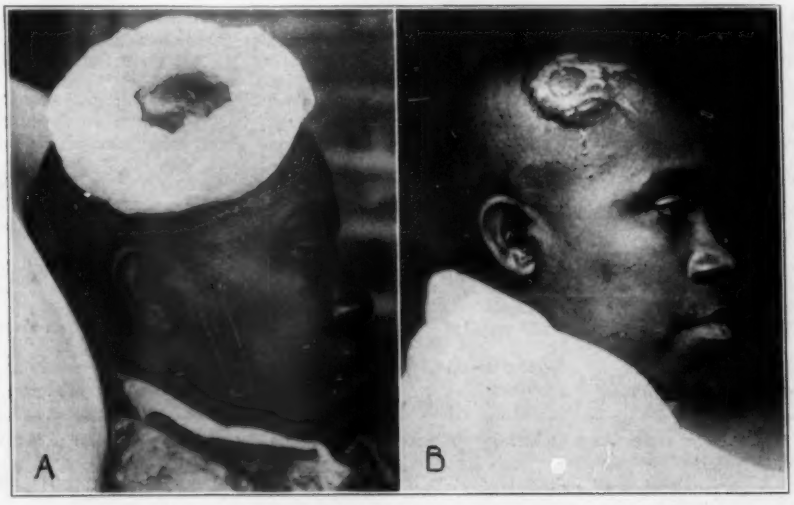

Fig. 16 (case 14).—*A*, protection of fungus by a doughnut pad of gauze. This patient was admitted to the hospital with a fungus following incomplete evacuation of an abscess. *B*, the fungus was diminished in size following complete evacuation of the abscess. The patient made a complete recovery from the abscess but had a residual paralysis of the left arm, and an occasional jacksonian attack on the left side.

frontal lobe. A large osteoplastic flap was made for exploration of the right frontal lobe. Through a small incision in the dura, a puncture of the frontal lobe was made after the base of the bone flap was fractured. A dense abscess wall was encountered at a depth of about 4.5 cm. The patient developed a large fungus (fig. 8 *A*) and had a stormy convalescence, but made a complete recovery (fig. 8 *B*). There were three small abscesses in case 5 (fig. 9, *A* and *B*), two of which were located at the first operation. The abscesses in this case arose from a chronic mastoid infection. The capsules appeared to be delicate and offered little resistance to the ventricle needle.

COMMENT

In a series of fourteen cases of chronic abscess of the brain, there were twelve recoveries, as shown in the accompanying table. The method of drainage was similar in every case; that is, the eye-end of a small, soft rubber catheter was used. The recoveries, except as indicated in the table, have been more or less complete in all of the nonfatal cases. An effort was made to evacuate the abscess completely at the primary operation, and an attempt was not made in any case to wash out the débris or to cleanse the cavity. The drainage following primary evacua-

Summary of Reported Cases

Case	Age	Sex	Operation	Location	Infection	Result
1	12	M	Yes	Right temporal	Staphylococcus	Recovered; no residuals
2	14	F	Yes	Right temporal	Staphylococcus	Recovered; no residulas
3	9	F	Yes (2)	Right temporal	Staphylococcus albus	Recovered; no residuals
4	14	M	Yes	Left temporal	Undetermined	Recovered; no residuals
5	38	M	Yes (2)	Left temporal (3)	Streptococcus	Recovered; no residuals
6*	43	M	Yes	Left temporal	Staphylococcus	Diffuse meningitis before operation; death
7	27	M	Yes (3)	Right frontal	Staphylococcus	Recovered; no residuals
8	19	M	Yes	Right frontal	Staphylococcus	Recovered; no residuals
9†	25	M	Yes	Right frontal (fungus)	Staphylococcus	Meningo-enceph.; death
10	25	M	Yes (3)	Left frontal	Staphylococcus	Recovered; no residuals
11	14	M	Yes	Left frontal	Staphylococcus aureus	Recovered; no residuals
12	1½	F	Yes	Right parietal	Staphylococcus aureus	Recovered; no residuals
13	8	M	Yes	Right parietal	Streptococcus	Recovered; hemiplegia
14	30	F	Yes (3)	Right parietal	Streptococcus	Recovered; hemiplegia

* First seen after onset of diffuse purulent meningitis, as determined by spinal puncture. Drainage of ruptured abscess.

† Admitted with large fungus from insecurely sutured decompression for supposed tumor of the brain.

tion was so slight in some of the cases that I feel that it might have been omitted. The tendency in the treatment for chronic subcortical abscess is to adopt simpler operative methods, and further progress may be expected in methods which, while securing the evacuation of the abscess, do not extensively traumatize and contaminate the brain tissues lying about the abscess wall.

Subcortical abscesses from gunshot wounds or those from infective embolic processes are not included in this series. In my experience, the mortality in these types of abscess has been high, and the patients have generally received little benefit from operations of any kind.

NEW METHODS OF ANASTOMOSIS OF THE COMMON BILE DUCT

AN EXPERIMENTAL STUDY *

GOLDER LEWIS McWHORTER, M.D., PH.D.
Assistant Clinical Professor of Surgery, Rush Medical College of the University of Chicago
CHICAGO

There is, at present, a general uncertainty about the satisfactory outcome of repair of the common bile duct. One may classify restoration of the bile passages into: (1) end to end suture of the ducts, (2) implantation of the bile ducts into the duodenum or stomach, (3) bridging a gap by a rubber tube or by foreign tissue grafts and (4) repair by pedunculated flaps from the stomach, duodenum, jejunum, gallbladder or skin.

End to end suture of a dilated part of the common duct over a buried rubber tube was performed by Doyen,[1] in 1892, following the removal of a large stone. Death resulted from hepatic insufficiency. Kehr [2] found it advisable to bring a rubber T tube out through the union for drainage. Jacobson [3] stated that complete closure of the duct at the suture line was undesirable, and he observed secondary stenosis in a number of cases at reoperation.

Mayo,[4] Bevan [5] and Moynihan [6] found it advisable to leave one side of the union open for a rubber tube. A gap of 2 or 3 cm. has been successfully sutured. Objections to this method are due to the large amount of fibrous tissue formed and further increased by the removal of the tube.

McArthur [7] completely sutured the common duct around a rubber catheter introduced into the duodenum. The catheter is held in place

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* Read before the Chicago Surgical Society, April 6, 1928.

1. Doyen, E.: Quelques operations sur le foie et les voies biliares, Arch. prov. de chir. **1**:149, 1892.
2. Kehr, H.: Chirurgie der Gallenwege, Neue Deutsche Chir. **8**:183, 1913.
3. Jacobson: Repair and Reconstruction of the Bile Ducts, Am. J. Obst. **70**:949, 1914.
4. Mayo, W. J.: Restoration of the Bile Passage after Serious Injury to the Common or Hepatic Ducts, Surg. Gynec. Obst. **22**:1 (Jan.) 1916.
5. Bevan, A. D.: Repair of the Common Duct, Surg. Clin. **4**:519 (June) 1920.
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by folding back the enlarged end on itself and by a chromic catgut suture. Duodenal stasis will pull the catheter out after a few weeks. This introduces a definite danger of ascending infection. However, Duval and Richard [8] inserted a rubber tube through the ampulla after all choledochotomies followed by primary suture of the duct.

Fowler [9] and others found reconstruction by end to end suture difficult without the use of a rubber tube. Walton [10] believed that without a tube leakage and stenosis are more likely to occur, and reported one case followed by attacks of jaundice.

Although the distal end of the common duct is frequently buried in adhesions and difficult to identify, W. J. Mayo [11] found it rarely contracted. McArthur stated that it should be utilized for reconstruction if at all possible in preference to implantation of the duct directly into the bowel.

Implantation of the bile ducts directly into the duodenum or stomach is frequently the easiest method. There is usually dilatation of the duct, but mobilization may be difficult because of adhesions. In 1891, Sprengel [12] successfully sutured the common duct to the duodenum. A number of patients have been successfully operated on, usually by suturing over a buried rubber tube (W. J. Mayo). McArthur and others, however, pointed out the greater danger of ascending infection and of stenosis than by suturing to the terminal end of the duct.

Recently, Judd and Parker [13] reviewed sixty-two cases of stenosis of the bile ducts in which reconstruction was inadvisable or impossible. Previous operations had been performed in all but one, including a cholecystectomy in forty cases. In fifty-four of these the bile ducts were sutured to the duodenum. Eight of the sixty-two patients died after a short time, seventeen were considered cured, sixteen, improved and nine in only fair health. Partial or complete contraction of the stoma occurred in seven of the forty-seven patients with hepaticoduodenostomy that required reoperation.

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8. Duval, P., and Richard, A.: Le drainage duodenal transvaterien par tube perdu dans la choledocotomie, J. de chir. **26**:129 (Aug.) 1925.

9. Fowler, R. S.: Repair of Common Bile Duct: A Case Report, Am. J. Surg. **37**:319 (Dec.) 1923.

10. Walton, A. J.: Reconstruction of the Bile Ducts, Proc. Roy. Soc. Med. **17**:24 (Nov.) 1923.

11. Mayo, W. J.: Surgery of the Hepatic and Common Bile Ducts, Lancet **1**:1299 (June 30) 1923.

12. Sprengel, quoted by Ginsburg and Speese: Ann. Surg. **65**:79, 1917.

13. Judd, E. S., and Parker, B. R.: Biliary Intestinal Anastomosis for Obstructive Jaundice: Analysis of 137 Consecutive Cases, Arch. Surg. **17**:1 (July) 1928.

One of my patients [14] has been well six years following a hepaticoduodenostomy. An absence of stenosis of the stoma was demonstrated a year later by filling of the bile ducts after a barium meal. Occasionally, after meals, on bending over at housework, the patient has had some discomfort in the right side.

In two experiments, Berg [15] showed that the gallbladder may empty satisfactorily after a choledochoduodenostomy.

Bridging a gap in the bile ducts by the use of a rubber tube covered with omentum, or by foreign tissue, such as fascia or vein, may occasionally be desirable or unavoidable. The lower end of the tube may be inserted, preferably into the terminal part of the common duct or directly into a new opening in the stomach or duodenum. While Doyen, McArthur and others used the rubber tube clinically, it was not until the experimental work of Sullivan [16] that it came into general use. He inserted the tube into the duodenum after the method of Witzel, but advised bringing it out through the ampulla if possible.

Lecène and Guadart-D'Allaines [17] prefered to use the rubber tube without attempting a direct suture of the ends. Muzeneek [18] experimentally concluded that bridging a defect with a rubber tube was satisfactory, but preferred to cover it with omentum or a segment of vein. In most instances, he found the inner surface of the fibrous wall covered with epithelium, although this did not prevent frequent stenosis. Experimentally, the insertion of a rubber tube for a long time did help to prevent stricture. Clinically, Weglowski [19] advised its use for treatment of a stricture at the ampulla.

Eliot [20] reviewed eighteen cases of reconstruction of the bile duct. In nine, the tube was inserted through the terminal duct and ampulla, while in the others it was introduced directly into the intestinal tract. He concluded that the use of the tube alone to develop granulation tissue was not as good as when this procedure was combined with a plastic

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14. McWhorter, G. L.: Congenital Cystic Dilatation of the Common Bile Duct, Arch. Surg. **8**:604 (March) 1924; Preventive Surgery of the Pancreas and Bile Ducts, Illinois M. J. **47**:128 (Feb.) 1925.

15. Berg, B. N.: Gall Bladder Function after Division of the Common Duct and Transplantation of the Proximal Segment, Surg. Gynec. Obst. **46**:464 (April) 1928.

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17. Lecène, P., and Guadart-D'Allaines, F.: J. de chir. **20**:237 (Sept.) 1922.

18. Muzeneek, P.: Experimentelle Untersuchungen zur Plastik der Gallengänge, Deutsche Ztschr. f. Chir. **195**:267, 1926.

19. Weglowski, R.: Presse méd. **34**:1124 (Sept. 4) 1926.

20. Eliot: The Repair and Reconstruction of the Hepatic and Common Bile Ducts, Tr. Am. Surg. A. **35**:222, 1917.

operation. Brewer [21] and a number of others reported stenosis following use of the tube to bridge a defect. Wilms,[22] C. H. Mayo,[23] Erdmann,[24] Coffey,[25] Feist,[26] Roith,[27] Gerlach [28] and others left the tube in place for long periods of time, or permanently. A few of these and others (Haggard,[29] Cope [30]) were successful, but in many instances the tube became filled with bile pigment and obstructed.

The chief dangers in the use of the rubber tube for reconstruction are leakage, ascending infection, occlusion and stenosis. The use of foreign tissue in place of, or in conjunction with, a rubber tube has not been satisfactory. Among tissues which have been used are arteries, veins, ureter, fascia and free transplants of peritoneum and omentum.

Horsley [31] found, experimentally, that constriction of the connective tissue which replaced the submucosa occurred even after it became lined with epithelium. When veins or other tissues having no immunity against bile were used, a large amount of scar tissue resulted, and he concluded that direct suture of the duct to the duodenum or stomach was more satisfactory.

Under circumstances in which it is impossible to mobilize the duct or the duodenum, a buried tube may be preferred (Phemister [32]).

Pedunculated flaps from the adjacent mucous-lined stomach, duodenum, jejunum, gallbladder or from the skin and fistulous tracts have been used to bridge defects in the bile ducts.

A cholecystenterostomy is frequently impossible because of the previous removal of the gallbladder or occlusion of the cystic duct.

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21. Brewer, G. E.: Hepatoduodenal Anastomosis, Ann. Surg. **51**:830, 1910.

22. Wilms: Deutsche Nat. u. Ärzte, 1911; Abstr. in Centralbl. f. Chir. **46**:1506, 1911.

23. Mayo, C. H., and Hendricks, W. A.: Internat. Clin. **1**:185 (March) 1926.

24. Erdmann, J. F.: Reconstruction of Bile Ducts, Ann. Surg. **67**:371, 1918.

25. Coffey, R. C.: Surgery of the Gall Tracts, Northwest. Med. **24**:326, 1925.

26. Feist, G. H.: Zur Gallengangsplastik mittels versenkter Drain, Beitr. z. klin. Chir. **135**:360, 1925.

27. Roith, O.: Ein Beitrag zum operativen Ersatz des Gallengänges, Deutsche Ztschr. f. Chir. **189**:73, 1925.

28. Gerlach, K.: Die Überbrückung operativer Choledochus Defekte mittels Gummrohrs, Deutsche Ztschr. f. Chir. **194**:195, 1926.

29. Haggard: Reconstruction of Bile Duct, Northwest Med. **24**:170 (April) 1925.

30. Cope, U. Z.: Intubation of the Common Bile Duct for Stricture, Lancet **2**:1169 (July 19) 1924.

31. Horsley, J. S.: Reconstruction of the Common Bile Duct, J. A. M. A. **71**: 1189 (Oct. 12) 1918.

32. Phemister, D. B.: Reconstruction of the Hepatic Duct, Surg. Clin. **1**:553 (June) 1917.

Haberland [33] advised suturing the gallbladder to the stomach instead of to the duodenum, because of less danger in kinking. Liebold [34] successfully split a contracted gallbladder and turned it over a defect in the common duct. Eliot believed that the mucosa of the gallbladder predisposes to calculi when used in the common duct. He advised against the inclusion of mucosa in flaps from adjacent viscera on account of the danger of ascending infection. A seromuscular flap from the stomach was used by Kehr.[35] Walton successfully turned up a flap from the duodenal wall and sutured it around a tube in the duct when it was impossible to identify the terminal end. Ginsburg and Speese [36] recommended this method with the duodenal flap turned downward. They reported failure in a patient operated on three times with the use of a tube alone. Mayo Robson [37] believed that a rubber tube was more satisfactory when combined with a plastic. Lamphear [38] turned up the end of the cut jejunum through the mesocolon and sutured it to the hepatic duct with a good result, in spite of a slight biliary fistula. Brandsburg [39] brought up a loop of jejunum subcutaneously and later connected it with the biliary fistula. He concluded that all plastic measures were dangerous. There have been a few reported cases of successful implantations of a fistulous bile tract into the duodenum.

The method of end to end suture of the common duct by leaving one segment open and bringing out a drainage or T tube (fig. 1, 1) has been widely accepted because the results were better than when drainage was not used. Failure is probably due to tension of the bile, separation of the ends, leakage of the bile or infection. Objections to leaving one side open for drainage are that about one third of the circumference of the lumen at the suture line is sacrificed with a certainty of further narrowing from fibrous tissue due to trauma of the tube and leakage of the bile. Diversion of the bile might better be obtained through a catheter, inserted above the anastomosis, or in the hepatic duct (fig. 1, 2).

Temporary diversion of the bile is important in permitting primary healing of a sutured or split duct, by placing it at physiologic rest.

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33. Haberland, H. F. O.: Arch. f. klin. Chir. **130**:492, 1924.

34. Liebold: Plastiche Deckung eines Choledochus-Defektes durch die Gallenblase, Zentralbl. f. Chir. **35**:501, 1908.

35. Kehr, H.: Ueber den plastischen Verschluss von Defekten der Choledochus Wand durch Netzstücke und durch Serosa-Muscularislappen aus Magen oder Gallenblase, Deutsche Ges. f. Chir. Verhandl. 31st Congress, 1902.

36. Ginsburg, N., and Speese, J.: Autogenous Fascial Reconstruction of the Bile Duct, Ann. Surg. **65**:79, 1917.

37. Robson, Mayo: Oxford Surgery, 1925, vol. 3.

38. Lamphear: Two Operations for Total Destruction of the Gall Tracts—One Fatal, One Successful, Surg. Gynec. & Obst. **8**:407, 1909.

39. Bradsburg, B.: Zur Frage über den plastischen Ersatz des Ductus Choledochus, Zentralbl. f. Chir. **53**:1877 (July 24) 1926.

This was accomplished experimentally by an original plan of placing either a plain or chromic catgut ligature about the common duct above the split or sutured area. Usually within a week or ten days the ligature was absorbed and the bile duct became patulous without evidence of scar. Clinically, a satisfactory diversion of the bile may result following suture of the common duct by a cholecystostomy.

In previous experiments in which I split the gallbladder from fundus to outlet, the cystic duct was ligated with catgut. There was a rapid

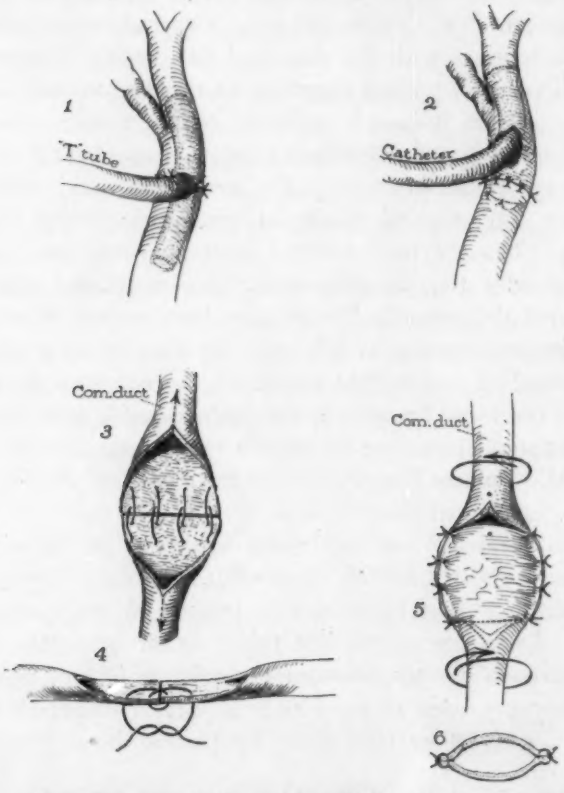

Fig. 1.—*1*, shows the commonly used technic of common duct suture; *2*, preferable method of utilizing complete circumference with diversion of bile; *3*, original method of "riband" end to end anastomosis after splitting the bile duct; *4*, a satisfactory approximation and tension suture, *5* and *6*, original method of overlapping the "riband" ends of the common duct, which doubles the lumen.

reconstruction and sealing of the gallbladder aided by adjacent viscera with a return of patency and function of the cystic duct.

There are several advantages obtained by splitting the common bile duct, which experimentally will accurately reconstruct itself if placed at physiologic rest. The duct will appear as a "riband" and may be more accurately explored for stones or strictured areas and resected.

After suture of the "riband" ends without closure of the sides (fig. 1, *3*), there cannot be any bile tension at the suture line.

R. Hamilton Russell [40] split open the urethra to any desired extent and followed this by excision of extensive strictures and suture of the "riband" ends. He excised the fibrous tissue, undercut the ends and united the ends with five catgut sutures. The urine was diverted above the suture line by fastening a rubber catheter into the bladder, and the split urethra was left wide open. No difficulty was observed in passing sounds or with a stricture.

In suture of the bile ducts, prevention of a stricture is of even greater importance, and one should therefore utilize a maximum of circumference of the duct ends and attempt to secure primary union with a minimum of fibrous tissue. Under unfavorable conditions, partial or complete separation, or a large amount of fibrous tissue may lead to narrowing of the lumen.

By an original method of suturing the overlapped split ends of the bile ducts, it has been possible to double the lumen in the important region of the anastomosis (fig. 1, *5* and *6*). This should prevent any tendency to late stricture.

In three experiments, in order to study healing and for comparison, the common bile duct was resected and the ends sutured without splitting. Because of the small size of the common bile duct, only three interrupted deeply caught sutures of fine silk were taken at equidistant points. Temporary diversion of the bile was obtained by a catgut ligature and drainage of the gallbladder.

The union was satisfactory in all three experiments (fig. 2, group III). In one instance (experiment 104), at the end of twenty-four hours the edges had become accurately agglutinated and sealed together. A second experiment (105) was terminated at forty-one days. There were no silk sutures in the lumen, which was good, but there was a slight narrowing, apparently due to a rather large amount of fibrous tissue about it. In this experiment, a free omental graft was used to cover the sutured area. In a third experiment (103), the animal died at the end of forty-seven days with a huge abscess of the right lobe of the liver. The fistula of the gallbladder had closed and the bile was draining normally through a good common duct. There were no sutures in the lumen. There was little fibrous tissue and no evident narrowing.

END TO END "RIBAND" ANASTOMOSIS

In thirteen experiments (table 1), the common duct was split nearly the entire length and a small segment resected. The two "riband" ends were sutured together with two, three or four deeply placed sutures of

40. Russell, R. Hamilton: The Treatment of Urethral Stricture by Excision, Brit. J. Surg. **2**:7, 1915; Med. J. Australia **1**:23 (March 22) 1919.

fine silk (fig. 1, 3). Catgut sutures were found to be too coarse, and after a few experiments, fine silk was used throughout.

In two experiments, the ends pulled apart. A satisfactory, deeply placed suture was devised in the form of a figure three with the ends

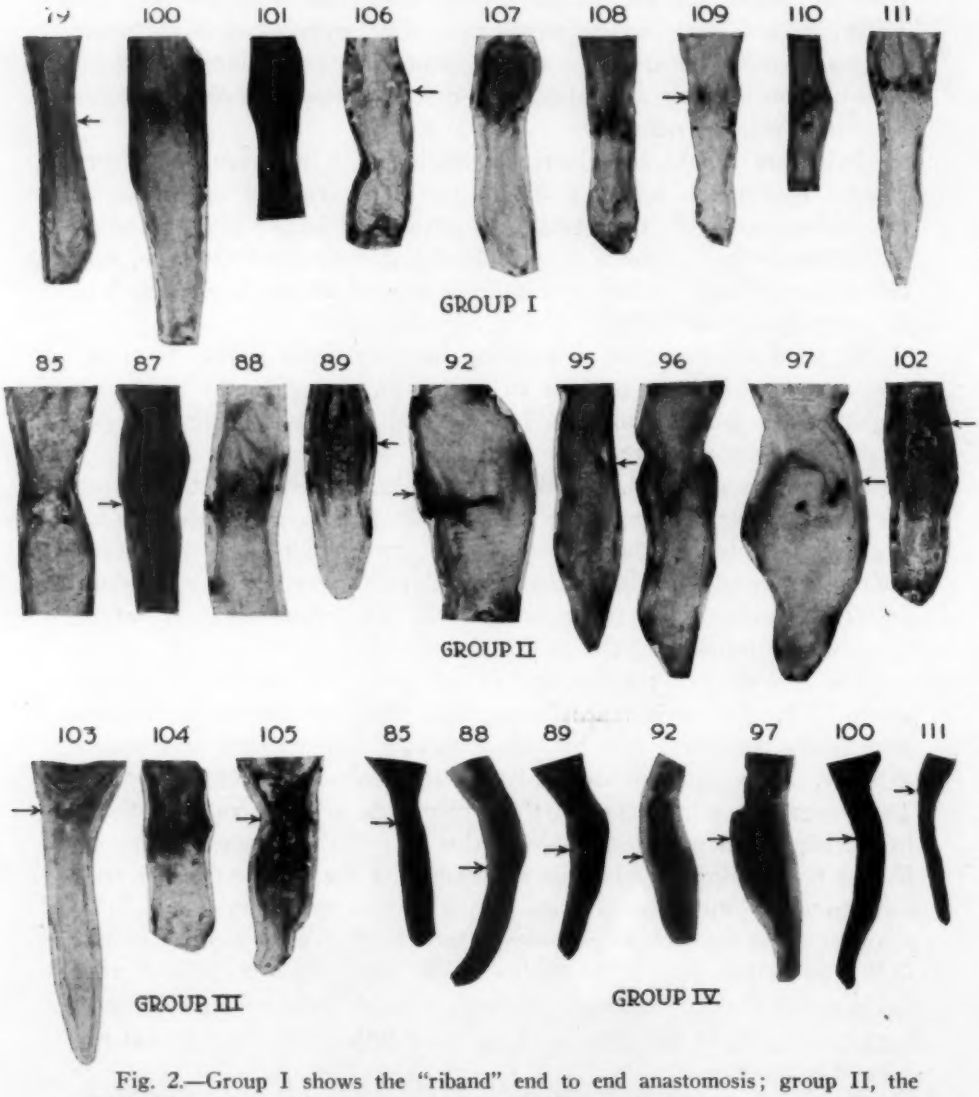

Fig. 2.—Group I shows the "riband" end to end anastomosis; group II, the overlapping side to side anastomosis; group III, circular end to end anastomosis, and group IV, roentgenograms showing the lumen. The arrows indicate the site of the anastomosis.

tied outside (fig. 1, *4*). The large loop extended from 3 to 4 mm. back from the edges, with the short loop approximating them.

In addition, the duodenum was sutured to the neck of the gallbladder in order to relieve the tension. In a third failure, the drained gallbladder had torn away from the abdominal wall and the sutured ends were edematous and partially pulled apart. Stricture would undoubtedly have been the late result.

Ten of the experiments were successful. Nine were terminated, many from distemper, after from seven to sixty-five days (fig. 2, group I). One animal was alive and in excellent health after eleven months. The intravenous injection of tetraiodophenolphthalein after four months showed the dye passing freely into the intestine, although the gallbladder failed to show.

The results in these experiments indicated a return of function to the common duct after a week or ten days, with closure of the gallbladder

TABLE 1.—*"Riband" End to End Anastomosis*

Experiment	Days' Duration		Results
79	65	Died	Good union; no sutures persisting
80	61	Died from peritonitis with bile leakage	Failure; ends separated
82	14	Died	Failure; ends separated
98	7	Died from peritonitis with bile leakage	Failure; ends separated
99		Alive 11 months	Normal function of duct
100	22	Killed	Good union; two sutures persisting
101	19	Died	Good union; no sutures persisting
106	17	Died	Good union; one suture persisting
107	24	Died	Good union; two sutures persisting
108	11	Died	Good union; three sutures persisting
109	16	Died	Good union; two sutures persisting
110	8	Died	Good union; three sutures persisting
111	18	Killed	Good union; three sutures persisting

fistula in two or three weeks. After a few weeks many of the silk sutures had passed into the duodenum, without bile precipitation, and it was then sometimes difficult to see the line of union. The occasional narrowing was evidently due to fibrous tissue about the anastomosis. Roentgenograms showed a good lumen (fig. 2, group IV, experiments 100 to 111).

OVERLAPPING "RIBAND" SIDE TO SIDE ANASTOMOSIS

In thirteen experiments (table 2), after simple division or resection of the duct, the ends were split for a short distance and overlapped so that the mucous surfaces were against each other (fig. 1, *5* and *6*). This doubled the size of the lumen and also permitted more than twice the length of edges available for suture than in the end to end suture. By splitting only one side of the duct, it was necessary to free and rotate each end 90 degrees so that the mucous surfaces were approximated. After division of the duct, however, each end may be split, one anteriorly and the other posteriorly, or on opposite lateral sides, so that

they will overlap without axial rotation. Unless desired for exploration, the duct should not be split above the point desired for overlapping. This permits a final closing suture from the center of each end to a point just back of the split portion on the other. (This is indicated by dots in figure 1, *5*.) The lateral edges of the overlapped ends were sutured with from two to four interrupted silk sutures.

Stenosis cannot result from contracture of the suture line, because the resulting union encircles only one half of the enlarged lumen at any point, and the elasticity of the wall of the normal duct opposite it should compensate for any contracture. Two failures were the result of pulling of the sutures. A third was the result of an evisceration and a fourth of an extensive wound infection with partial separation of the ends.

TABLE 2.—*"Overlapping" Side to Side Anastomosis*

Experiment	Days' Duration		Results
85	43	Died	Good union; four sutures persisting
86	9	Died of evisceration	Failure; ends of duct separated
87	8	Killed	Good union; one suture persisting
88	53	Killed	Good union; three sutures persisting
89	47	Died	Good union; no sutures persisting
90	4	Died of evisceration	Failure; ends separated partially
91	12	Died	Failure; ends separated
92	75	Killed	Good union; two sutures persisting
93	14	Died of bile leak	Failure; ends separated
95	54	Killed	Good union; two sutures persisting
96	6	Killed	Good union; all sutures persisting
97	35	Killed	Good union; three sutures persisting; small chronic abscess near duct
102	18	Died	Good union; all sutures persisting

Nine experiments were successful (fig. 2, group 2). They were terminated in from eight to sixty-five days. The common duct was found to be functioning after from eight to ten days. In one experiment, at the end of six days the anastomosis was apparently sealed. A bile leak did not occur in the successful experiments, as the common duct was sealed by the time the catgut ligature was absorbed, from the eighth to the twelfth day. The specimens usually showed a small amount of fibrous tissue about a faint slightly oblique scar with an occasional persisting suture. In some experiments, an evident widening of the lumen was present in the region of the overlapped and sutured ends. Roentgenograms of the healed ducts showed normal and some definitely widened lumens (fig. 2, group IV; experiments 85, 88, 89, 92 and 97).

In experiment 97, after thirty-five days a thick walled off abscess sac, 1.5 cm. in diameter, was found adjacent to the common duct but without any disturbance of its function. A microscopic examination showed a thick fibrous wall lined by granulation tissue and a normal liver.

Microscopic examinations were made of the common duct through the line of anastomosis in experiments 92 and 95. In both experiments, there was accurate healing of the wall with only a small amount of fibrous tissue externally.

Microscopic examination was made of the liver following the anastomosis in experiments 87, 88, 92, 95, 97, 100 and 105. There was neither evidence of inflammation nor of abnormal changes.

The splitting of the common duct and freeing of the ends by blunt dissection apparently did not result in a disturbance of the blood supply. However, care should be used to avoid hemorrhage.

In the experiments with splitting alone or when followed by resection and an anastomosis, there was usually a faint linear scar with slight evidence of narrowing. In no instance were the sides of the split duct sutured, but they were covered by a thin piece of omentum or peritoneum which appeared to help seal the edges of the duct together. The healed split edges some distance away from the anastomosis showed little if any gross fibrous tissue. In three experiments, after two or three weeks with normal function of the duct, it was possible by fluid distention to separate the split edges some distance above the anastomosis with diffusion beneath, but not rupture of the adherent peritoneum. In the healing of the experimentally split gallbladder it was observed that usually either the omentum, duodenum or liver aided in sealing the opening.

If one attempts to get a bile-tight lumen by suturing the split common duct, it may be necessary to invert the edges and narrow the lumen. With a large or dilated and thickened duct, suture is often desirable. Clinically, following drainage I have observed leakage of bile. It would also seem from experiments on animals (80 and 98) that abdominal drainage should be used. Bile tension which may interfere with healing may be relieved by proximal diversion of the bile through a tube or by a cholecystostomy. A buried tube introduced through the ampulla may also relieve tension, but there is greater danger of infection.

Following diversion of the bile above a split or sutured bile duct, there is no retention at the sutured area. The mucosal surfaces of the lumen cannot become adherent, and their edges promptly become sealed together when covered with a peritoneal surface. Usually, only a few adhesions were found in the abdomen. Fibrous tissue is minimized by an accurate firm anastomosis with an absence of bile, infection, traction or foreign material, such as rubber tubes.

In animals, fine silk was found preferable to catgut for suturing the common duct. Deep, firm sutures were necessary. Later they passed out through the ampulla without trouble. Clinically, chromic catgut is preferred because of less danger in stone formation (Kehr).

When the results of the various methods of suturing were compared, it was observed that overlapping of the split ends of the common duct resulted in a widening at the junction, while that of the circular and "riband" end to end suture ended in an occasional slight narrowing of the lumen.

It is probable that none of these methods would result in stricture formation with primary union or in the absence of surrounding excessive fibrous tissue. In attempting to suture the end of a thin common duct to a flap of duodenum much greater difficulty is present experimentally than with the suture of the two ends of the duct, and infection cannot be avoided. Clinically, there may be distinct advantages obtained in the exposure and exploration permitted by splitting the bile duct.

CONCLUSIONS

1. After operations on the common duct diversion of the bile is an aid to primary union.

2. The bile ducts become permeable after ligation with catgut.

3. Splitting of the bile ducts without suture resulted in rapid reconstruction.

4. Microscopic evidence of inflammation was not observed in the liver or common duct after successful suture.

5. In end to end union, either with or without splitting of the common duct, the entire circumference should be sutured.

6. The overlapping method of anastomosis with the "riband" common duct gives a larger lumen and firmer union than the end to end method.

122 South Michigan Avenue.

COLLOID CARCINOMA OF THE COLON AND RECTUM *

FRED W. RANKIN, M.D.
AND
CHARLES L. CHUMLEY, M.D.
Fellow in Surgery, The Mayo Foundation
ROCHESTER, MINN.

The origin and significance of the gelatinous material found in tumors designated colloid carcinoma has long been a subject of interest. Colloid has been described as a product of degeneration taking place in a tumor; it has probably been derived either from epithelial or connective tissue, or both. It is now agreed that it is secretory in origin and derived from the epithelial cells of tumors arising from any of the glandular epithelial structures. Accordingly, Broders [1] has defined colloid carcinoma as adenocarcinoma with a tendency to differentiate to the extent that a mucus-like substance is produced, and Robertson [2] affirmed that a colloid carcinoma is so-called because it is really a collection of mucus large enough to be appreciable to the eye.

A distinction between mucus and colloid is no longer maintained; they are usually considered identical substances. Wells,[3] however, made such a distinction. He submitted a definite chemical composition for mucus, and described epithelial and connective tissue types of mucus. He expressed the belief that the term "colloid" has an indefinite meaning and that it is merely morphologically and macroscopically descriptive of certain products of cellular activity or degeneration which have nothing in common, except that they form a thick gluelike or gelatinous substance. He believed that one should recognize as the usual colloid substances: (1) thyroid colloid, (2) mucin, (3) pseudomucin and (4) simple proteins.

Certain pathologists believe that colloid and mucoid carcinomas are degenerations of other types of tumors. These two types are believed by some to be separate and distinct lesions that can be distinguished on a chemical basis. To quote Delafield and Prudden: [4]

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* From the Division of Surgery, The Mayo Clinic.

1. Broders, A. C.: The Grading of Carcinoma, Minnesota Med. **8**:726, 1925.

2. Robertson, H. E.: Unusual Features of Carcinoma, Minnesota Med. **8**:1, 1925.

3. Wells, H. G.: Chemical Pathology; Being a Discussion of General Pathology From the Standpoint of the Chemical Processes Involved, Philadelphia, W. B. Saunders Company, ed. 5, 1925, p. 790.

4. Delafield, Francis, and Prudden, T. M.: A textbook of Pathology, with a Final Section on Postmortem Examinations and the Methods of Preserving and Examining Diseased Tissues, New York, William Wood & Company, ed. 13, 1925, p. 54.

The simplicity of the earlier distinction has, however, not been justified by more modern research, so that the separation of mucus and colloid degenerations along the old lines does not express very definite chemical knowledge.

Miles [5] stated that colloid carcinoma is merely a degenerative stage of the other varieties, both the epithelial and the connective tissue elements undergoing mucinoid change. Boyd [6] was also of the belief that these tumors were the result of colloid degeneration, and that the term colloid carcinoma is undesirable, since it gives rise to the belief that this is a special variety of carcinoma. McFarland [7] suggested that one proof that colloid carcinoma is a distinct and separate variety and not a common form degenerated seems to be found in the regularity with which the gelatinous structure is repeated in the secondary tumors.

There seems to be little doubt that colloid, in tumors of the gastrointestinal tract, is a product of secretion of the epithelial cells of these tumors. The manner in which mucus is formed in these tumors, especially those of the signet-ring type, has been described by McFarland as follows:

Ordinarily, when columnar epithelial cells form mucin, it appears in the form of a vacuole in the distal two-thirds of their substance, from which it is discharged upon the surface, and there swells to form the structureless mucus. But in carcinoma the cells no longer maintain their relation to the surface, but are in the depths of the tissue; they also no longer retain their polarity, and as anaplasia more and more disturbs their morphology, they assume more nearly the spherical form. Under these circumstances, any mucin formed in their substance is more centrally situated, and the cells become large spheres in which the increasing droplet of mucin swells more and more until it gives each cell the form of a 'seal ring.' As the cells eventually rupture and liberate their contents, the mucin absorbs water and swells to form mucus, which distributes among the cells, widely separating them.

While studying the significance of mucus-forming cells in carcinoma of the colon and rectum, Ochsenhirt [8] made sections from 188 of these growths. The sections were stained with mucicarmine which stains mucus and mucus-containing tissue red, in contrast to other tissues which are stained blue. He then graded the sections according to Broders' method of grading malignancy and devised a method for the purpose of grading the number of mucus-forming cells present. He concluded that the presence of mucus in carcinoma of the colon and

5. Miles, W. E.: Cancer of the Rectum, London, Harrison and Sons, 1926, p. 72.

6. Boyd, William: Surgical Pathology, Philadelphia, W. B. Saunders Company, 1925, p. 200.

7. McFarland, Joseph: Surgical Pathology, Philadelphia, P. Blakiston's Son & Company, 1924, p. 377.

8. Ochsenhirt, N. C.: The Significance of Mucus-forming Cells in Carcinoma of the Large Intestine and Rectum, Surg. Gynec. Obst. **47**:32, 1928.

rectum is the result of partial differentiation of the carcinoma cells. The more malignant the carcinoma, or the less the extent of differentiation, the less numerous the mucus-secreting cells and vice versa. The number of mucus-secreting cells in the carcinoma is inversely proportional to the grade of malignancy. In Ochsenhirt's series were twenty-two colloid carcinomas which were exceptions to the foregoing conclusions. In the less malignant types of colloid carcinoma the number of mucus-forming cells did not seem to be increased, as compared to those found in an adenocarcinoma of the same grade; however, there was an increase in mucus, as was indicated by the staining. This was probably due to increased activity on the part of the mucus-forming cells. In the higher grades of colloid carcinomas, he found mucus greatly in excess. In the signet-ring type, goblet cells or cells resembling the typical mucus-secreting cells were not found, yet almost all the cells were of the signet-ring type and were filled with mucus.

Parham [9] studied a large series of colloid carcinomas of the entire gastro-intestinal tract and came to the following conclusions: 1. The epithelial cells possess an uncontrolled function of secreting a mucinous substance and its accumulation is often destructive to the carcinoma cells. 2. The formation of mucus is a sign of functional differentiation of the carcinoma cells, corresponding to the morphologic differentiation in carcinomas with cells of the acinar or columnar types. 3. The mucus-forming characteristic may be possessed by cells either showing or not showing other signs of differentiation. 4. Colloid carcinoma is usually slow of growth and late to metastasize to lymph nodes and to other organs; it often grows by permeation and may cause extensive thickening of the wall of the affected organ. 5. Local lymph nodes are often involved long before metastasis has reached distant glands; although histologically less malignant on account of permeation of adjacent tissues, it is particularly difficult to eradicate; death is often delayed but the eventual mortality is greater than in other types of carcinoma. 6. Recurrence is often limited entirely to the site of origin.

Hayes [10] dissected the regional lymph nodes in 100 cases of carcinoma of the large intestine; 16 of these were of the colloid variety. He divided the cases into two groups of eight each; those with and those without involvement of the regional nodes. There were no recurrences in the group without lymphatic involvement; there were recurrences in six of the eight with lymphatic involvement.

From Jan. 1, 1907, to Jan. 1, 1927, operation was performed in 3,202 cases of carcinoma of the colon and rectum at The Mayo Clinic,

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9. Parham, Duncan: Colloid Carcinoma, Ann. Surg. **77**:90, 1923.

10. Hayes, J. M.: The Involvement of the Lymph Glands in Carcinoma of the Large Intestine, Minnesota Med. **4**:653, 1921.

and 158 of these were of the colloid variety. The distribution of the latter was as follows: cecum and ascending colon, forty-two (26.5 per cent); transverse colon, twenty-six (16.4 per cent); descending colon, three (1.8 per cent); sigmoid, fourteen (8.8 per cent), and rectosigmoid and rectum, seventy-three (46.2 per cent).

Of the 158 patients with colloid carcinoma on whom operation was performed, resection was performed in only 122 (70.8 per cent), palliative operations were performed in 12 (9.8 per cent) and exploration only in 24. All of the patients whose condition was inoperable are dead. The average duration of life was seven and a half months; the longest period of life was twenty-four months.

The clinical histories of the 158 patients with colloid carcinomas have been studied. Sections were taken from each operative specimen, fixed and stained with mucicarmine after the technic of Ochsenhirt. Broders then graded each section microscopically, according to his method of grading malignancy, and he also graded the mucus according to the method devised by Ochsenhirt.

INCIDENCE

The incidence of colloid carcinoma of the colon and rectum varies greatly with various observers. Parham found that 5.5 per cent of carcinomas of the rectum and rectosigmoid and as high as 22.2 per cent of those of the cecum and ascending colon were of the colloid variety. Korte [11] found colloid carcinoma in 11.7 per cent of seventy-two tumors of the colon. Zinner [12] noted the colloid type in 41 per cent of 123 cases in which carcinoma of the rectum occurred. In this series, the incidence was 4.93 per cent. Stinson [13] reported the incidence of colloid carcinoma in the stomach as 5.09 per cent, and Parham reported this condition in the breast and ovary as 1.12 per cent and 1.1 per cent, respectively. According to Robertson, a high percentage of tumors arising from glandular epithelial structures will, on microscopic examination, show mucus formation. Broders [14] recently studied 600 specimens from patients with carcinoma of the rectum microscopically and noted colloid formation in 16 per cent.

SEX AND AGE

In the series were 115 males (72.7 per cent) and 43 females (27.2 per cent). The average age was 50.2 years; the oldest patient was 75 and the youngest 16.

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11. Korte, quoted by Ewing, James: Neoplastic Diseases, a Textbook on Tumors, Philadelphia, W. B. Saunders Company, ed. 3, 1928, p. 714.

12. Zinner, quoted by Ewing.

13. Stinson, J. W.: Colloid Carcinoma of the Stomach, Surg. Gynec. Obst. **46**:180, 1928.

14. Broders, A. C.: Personal communication to the authors.

SYMPTOMS

In the cases of tumors of the rectum and of the rectosigmoid, rectal bleeding was the most common symptom, being present in fifty-two (71 per cent) of the cases. The quantity of blood varied from small amounts to frank hemorrhage in four cases (5.3 per cent). Loss of weight was noted in fifty (68.6 per cent) of the cases. Constipation was present in twenty-nine (39.1 per cent) and diarrhea in eighteen (24.6 per cent). Rectal pain was present in twenty-one (28.7 per cent) of the cases. In only three (4.1 per cent) were symptoms referable to obstruction.

In the cases of tumors of the colon, blood was noted in the stool in ten (11.7 per cent), and in only one was there hemorrhage from the bowel. In twenty-four cases (28.2 per cent), there was constipation

Fig. 1.—Colloid carcinoma graded 1 with mucus formation graded 2; × 75.

and in only five (5.8 per cent) diarrhea. Loss of weight was a common symptom, being noted in sixty-four cases (75.2 per cent). Abdominal pain of some degree was present in fifty-seven cases (67 per cent). In fourteen cases (16.4 per cent), the patients were aware of abdominal tumor, while in forty-six (54.1 per cent) a tumor was palpated during the course of an examination. The average duration of symptoms before admission was twelve and a half months.

GRADING OF MALIGNANCY AND AMOUNT OF MUCUS

By arranging these cases in four groups according to the grades of malignancy alone, thirty-five (28.7 per cent) were found to be graded 1 (figs. 1 and 2), forty-nine (40.1 per cent) were graded 2 (fig. 3), twenty-six (21.3 per cent) were graded 3 (fig. 4), and twelve (9.8 per cent) were graded 4 (fig. 5). In a similar manner, the cases were

grouped according to the grading of the amount of mucus; twelve (9.8 per cent) were graded 1 (fig. 4), twenty-one (17.2 per cent) were graded 2 (fig. 1), thirty-five (28.7 per cent) were graded 3 (fig. 3), and fifty-four (44.2 per cent) were graded 4 (figs. 2 and 5). A comparison of the two groupings (table 1) shows that the highest percentage

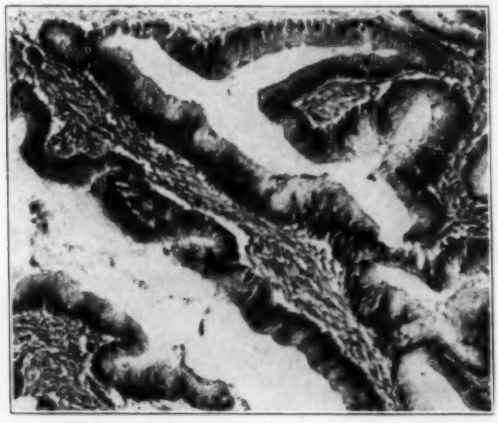

Fig. 2.—Colloid carcinoma graded 1 with mucus formation graded 4; × 75.

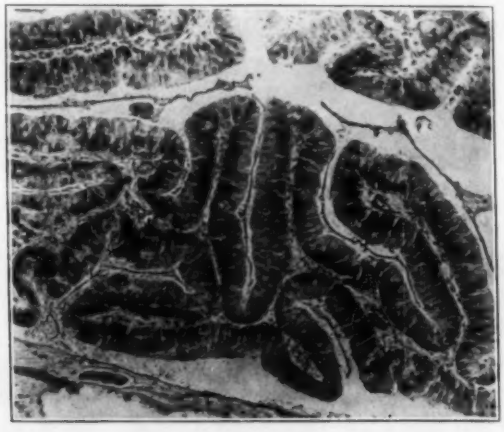

Fig. 3.—Colloid carcinoma graded 2 with mucus formation graded 3; × 75.

of cases falls in the lower grades of malignancy, but in the higher grades of the amount of mucus.

A comparison of the grading of the amount of mucus in relation to the grade of malignancy (table 2) shows that in the highest percentage of cases in each grade of malignancy mucus was graded 4. Mucus graded 3 ranked second in all grades of malignancy except grade 4; in 25 per cent of the cases in this group mucus was graded 1.

A comparison of the grade of malignancy in relation to grading of mucus is shown in table 3. Cases in which mucus was graded 1 are distributed fairly equally among the grades of malignancy, but in the cases of mucus graded 2, malignancy was graded 2 in 76.1 per cent. In cases of mucus graded 3, the highest percentage of cases was found to be in the groups in which malignancy was graded 1 and 2; the same distribution was found among cases in which mucus was graded 4.

TABLE 1.—*Grade of Malignancy and of Mucus in 122 Patients with Colloid Carcinomas*

	Grade 1		Grade 2		Grade 3		Grade 4	
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Malignancy	35	28.7	49	40.1	26	21.3	12	9.8
Mucus	12	9.8	21	17.2	35	28.7	54	44.2

TABLE 2.— *Grade of Mucus in Relation to Grade of Malignancy*

Malignancy			Mucus							
			Grade 1		Grade 2		Grade 3		Grade 4	
Grade	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
1	35	28.7	2	5.7	4	11.4	11	31.5	18	51.4
2	49	40.1	4	8.0	15	30.0	13	26.5	17	34.7
3	26	21.3	3	11.5	..	....	11	42.3	12	46.1
4	12	9.8	3	25.0	1	8.3	1	8.3	7	58.3

TABLE 3.—*Grade of Malignancy in Relation to Grade of Mucus*

Mucus			Malignancy							
			Grade 1		Grade 2		Grade 3		Grade 4	
Grade	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
1	12	9.8	2	16.6	4	33.3	3	25.0	3	25.0
2	21	17.2	4	19.0	16	76.1	..	....	1	4.7
3	35	28.7	11	31.5	13	37.1	10	28.5	1	2.8
4	54	44.2	18	33.3	17	31.4	12	22.2	7	12.9

Thus, it is shown that in colloid carcinoma of the lower grades of malignancy (graded 1, 2 and 3) there is a tendency for the grading of the amount of mucus present to be inversely proportional to the grade of malignancy, as was shown by Ochsenhirt in cases with adenocarcinoma of the colon and rectum. In the highest grade of malignancy (grade 4), 58.3 per cent showed the highest grade of mucus formation, which is hard to explain since mucus, according to Ochsenhirt, is a sign of partial differentiation, and according to Parham, colloid in colloid carcinomas is a result of functional differentiation. Robertson called attention to the mucus-secreting epithelial cell and stated that it is the

least differentiated and that growths from these cells retain with "exceeding tenacity" this particular characteristic. It may be seen how the cells of carcinoma graded 4 which have retained their mucus-secreting power will necessarily, by virtue of their rapid growth and division, produce a large amount of mucus. The cells of these growths are

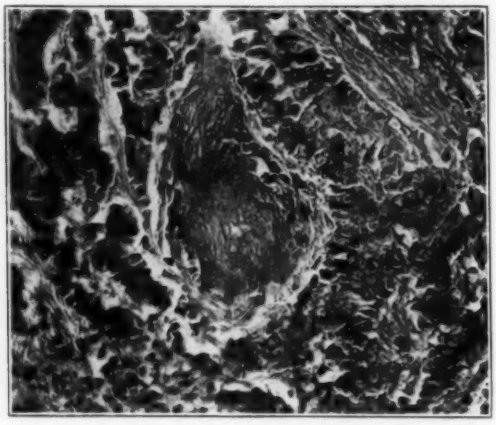

Fig. 4.—Colloid carcinoma graded 3 with mucus formation graded 1; × 75.

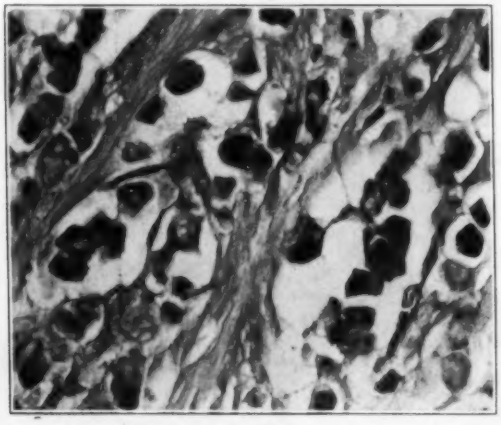

Fig. 5.—Colloid carcinoma graded 4 with mucus formation graded 4.. Cells are of the signet-ring type; × 350.

usually of the signet-ring type. On the other hand, if the cells have lost most of their mucus-secreting property, they will produce less mucus, although they grow rapidly. Why the cells of colloid carcinoma, which do not show any morphologic differentiation, should retain their functional activity to a high degree in one case and almost lose it in another is a question that has not been answered.

LYMPHATIC INVOLVEMENT, RECURRENCE AND MORTALITY

In the series of 122 patients, 23 died from the operation; of the remaining 99 patients, 92 have been traced. Sixty (65.2 per cent) are dead and thirty-two (35.7 per cent) are living. Lymphatic involvement occurred in seventy-one (58.1 per cent) of the cases in which resection was performed. Of the ninety-two patients traced, fifty-one had lymphatic involvement and of these forty-two (82.3 per cent) had recurrences, while only eighteen (43.9 per cent) of forty-one patients without lymphatic involvement were found to have had recurrence. All of the sixty patients who had had recurrences were dead, the average duration of life after operation being 20.2 months.

TABLE 4.—*Grade of Malignancy and Duration of Life in Cases of Recurrence*

	Malignancy							
	Grade 1		Grade 2		Grade 3		Grade 4	
Length of Life	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Less than three years	9	60.0	17	80.9	15	93.7	9	90.0
Three years or more	6	40.0	4	19.0	1	6.2	1	10.0
Four years or more	4	26.6	2	9.5	1	6.2		
Five years or more	1	6.6	1	4.7	1	6.2		

TABLE 5.—*Grade of Mucus and Duration of Life in Cases of Recurrence*

	Mucus							
	Grade 1		Grade 2		Grade 3		Grade 4	
Length of Life	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Less than three years	7	87.5	4	80	14	73.8	25	83.3
Three years or more	1	12.5	1	20	5	26.4	5	16.6
Four years or more	..	....	1	20	4	21.0	2	6.6
Five years or more	..	....	1	20	2	10.5		

The influence of the grade of malignancy on the duration of life of patients dying of recurrence is shown in table 4. In 40 per cent of the cases graded 1, the patients lived more than three years. In 19 per cent of the cases graded 2, in 6.2 per cent of cases graded 3 and in 10 per cent of those graded 4, the patients lived as long as three years. Table 5 shows the influence of the grade of mucus on the same group of cases, and the highest percentage of patients living three years or more are found in cases in which mucus was graded 2 and 3. Table 6 shows a comparison of the grades of mucus and malignancy in the resected tumors in twelve cases in which the patients are alive and well five years or more after operation; in eleven of these cases malignancy was graded 1 and 2, and mucus was graded 2 and 3. It may be seen from the foregoing that the best prognosis in colloid carcinoma of the colon and rectum is to be expected in cases in which malignancy is

graded either 1 or 2. It may also be noted that the grading of mucus is of value as a prognostic factor. When the grading of the amount of mucus is 2 or 3, the chances for postoperative longevity seem to be greatest. This is more apparent when one recalls (table 3) that 95.1 per cent of the tumors in this series graded 2 for mucus, and 68.6 per cent of those graded 3 for mucus were found to be graded either 1 or 2 for malignancy. The influence of the grades of malignancy and mucus on

TABLE 6.—*Grade of Malignancy and Mucus in Twelve Cases in Which the Patients Were Alive and Well More than Five Years After Resection*

	Grade 1		Grade 2		Grade 3		Grade 4	
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Malignancy	5	41.6	6	50.0	1	8.3		
Mucus	..	....	4	33.3	7	58.3	1	8.3

TABLE 7.—*Grade of Malignancy and Mucus Compared with Lymphatic Involvement, Recurrence and Mortality*

Mucus		Lymphatic Involvement		Recurrence		Living		Dead	
Grade	Cases	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Malignancy, Grade 1									
1	2	..	....	1	50.0	1	50.0	1	50.0
2	4	1	25.0	..	....	4	100.0	..	....
3	9	1	11.1	6	66.6	3	33.3	6	66.6
4	12	8	66.6	7	58.3	5	41.7	7	58.3
Malignancy, Grade 2									
1	3	1	33.3	1	33.3	2	66.6	1	33.3
2	10	6	60.0	5	50.0	5	50.0	5	50.0
3	10	5	50.0	4	40.0	6	60.0	4	40.0
4	13	6	46.1	10	76.9	3	23.1	10	76.9
Malignancy, Grade 3									
1	2	2	100.0	2	100.0	..	....	2	100.0
2	..	..	.....	..	.....	..	....	..	.....
3	9	6	66.6	7	77.7	2	22.3	7	77.7
4	8	8	100.0	7	87.5	1	12.5	7	87.5
Malignancy, Grade 4									
1	3	..	.....	3	100.0	..	....	3	100.0
2	..	..	.....	..	.....	..	....	..	.....
3	1	1	100.0	1	100.0	..	....	1	100.0
4	6	6	100.0	6	100.0	..	....	6	100.0

lymphatic involvement, recurrence and mortality in this series is shown in table 7.

Three patients in the series were apparently well for a long time, and then symptoms developed which caused them to return to the clinic. In each a second tumor was found in an entirely different segment of the colon than the first growth which had been resected a number of years before. One of the patients lived five years and ten months after resection of the transverse colon, and was then subjected to a second operation for tumor of the cecum. Another patient returned six years

and four months after operation for colloid carcinoma of the descending colon, and a similar growth was removed from the transverse colon. A third patient returned six years and nine months after resection of the cecum and ascending colon and was found to have a lesion of the sigmoid. Another patient in the series was subjected to three resections of the transverse colon over a period of about six years. All of these tumors were of the colloid variety, and with one exception the gradings of malignancy and mucus were the same in the primary and secondary tumors of each case. The fourth case was undoubtedly one of recurrence, but in the other cases one could not be sure whether the second tumor had its origin in the first tumor or whether it was an independent growth.

SUMMARY AND CONCLUSIONS

1. Colloid carcinoma occurred in about 5 per cent, 4.9 per cent, of the 3,202 cases of carcinoma of the colon and rectum.

2. A high percentage of colloid carcinomas of the colon and rectum were of a low grade of malignancy, but showed a high grade of mucus formation. In the lower grades of malignancy, there was a tendency for the amount of mucus present to be inversely proportional to the grade of malignancy.

3. The grading of the amount of mucus present in colloid carcinoma is of value as a prognostic factor; grades 2 and 3 offer the best chance for postoperative longevity.

4. If the lymph nodes are involved in colloid carcinoma, the prognosis is unfavorable regardless of the grade of malignancy or the amount of mucus present.

GASTRECTOMY *

JOHN M. T. FINNEY, M.D.
AND
WILLIAM FRANCIS RIENHOFF, JR., M.D.
BALTIMORE

After a fairly exhaustive review of the literature on subtotal and total gastrectomy, it would appear that credit for having first conceived the idea of resection operations on the stomach must be given to "a certain famous professor, highly respected and renowned among the medical profession of Philadelphia." This statement is based on the authority of Merrem of Giessen who, in 1810, contributed a monograph entitled "Certain Surgical Operations and Experiments on Animals Illustrated by Facts." In his monograph, Merrem referred to this famous Philadelphia professor without mentioning his name or giving the source of his information. Having consulted all available authorities, including the Surgeon-General's library in Washington, the libraries of The College of Physicians in Philadelphia and the New York Academy of Medicine, we have not as yet found any other record of this epoch-making work by this distinguished surgeon. The idea of this operation was suggested to this "famous Philadelphia professor," so Merrem stated, by "the terrible sufferings of his beloved friend, Dr. Middleton." Having tried everything else without avail, he suggested extirpation of the pylorus in order to relieve the obstruction caused by the disease. The operation appeared to involve such serious danger that the unknown physician would not venture to undertake it. Merrem went on to state that "two years earlier this Philadelphia doctor had had experiments made on several dogs," in which resection of the pylorus had been done, but all of them had "terminated fatally." The record of these experiments appears to be the first authoritative reference that can be found in the literature to resection operations on the stomach which were deliberately undertaken. To this unknown Philadelphia surgeon, therefore, must be given credit for having been the pioneer in this particular field. After a thorough study of all available data and by comparison of dates, etc., we feel that there can be no reasonable doubt but that the "famous Philadelphia surgeon" was none other than John Jones, a native of Philadelphia, where he lived the last few years of his life. But he was also the first professor of surgery in King's College, New York, afterward the College of Physicians and Surgeons and now Columbia University.

Stimulated by these experiments, Merrem himself later performed a number of resection operations on the stomach in dogs. Basing his

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* From the Department of Surgery of the Johns Hopkins University.

opinion on these experiments, he was led to state "that extirpation of the pylorus can be accomplished with happy results seems to be confirmed by these experiments; nevertheless, experience has shown that it is a very difficult operation." It is interesting to note that the criteria given by him for performing the operation, nearly a century and a quarter ago would be equally acceptable today. They are as follows:

1. If the patient seems the sure prey of death after having been sick for a long time, and after every other remedy has been tried to no avail.

2. If we find by placing the fingers on the right region of the stomach an unmistakable hardening.

3. If a short time after eating the patient suffers from obstruction of the bowels and chronic vomiting.

This thesis of Merrem did not, however, make much impression on the surgical profession, and it was not until 1876, sixty-six years later and almost a century after John Jones' work, that Gussenbauer and von Winiwarter reported their observations on dogs in which partial resection of the stomach had been done. Their publication marked an epoch in the progress of gastric surgery, as it demonstrated that dogs could live indefinitely after resection of considerable portions of the stomach. Their observations also contributed materially to the development of a proper technic for the operation. Two years later, in 1878, Kaiser reported from Czerny's clinic his work along the same line. Of five animals in which almost the entire stomach had been resected, one dog survived for five years and was then killed and the specimen examined by Ludwig, the famous physiologist. It showed that a small piece of stomach wall remained at the cardiac end, "which enclosed a spherical cavity filled with food." Physiologically, the dog appeared quite healthy, and the digestion seemed not to be impaired in any way.

At a meeting of the Royal Society of Physicians in Vienna, in 1898, Albert, in the course of the discussion on a paper by Frank on resection of the pylorus, stated that as early as 1880 he had advocated the idea of total extirpation of the stomach. He had developed a definite procedure on the cadaver, which he described. He further stated that he had tried it on a dog and had also planned to perform it on a patient, but that on opening the abdomen, he had found the tumor so firmly fixed that he was compelled to give up the operation. In this discussion, he also stated that in the early eighties Professor Nicoladoni had conceived the idea of total resection of the stomach in man, but he did not give any reference.

In this connection, it is of historical interest to note Billroth's famous prophecy made in 1877. It was while speaking of the operation of gastrorrhaphy, the closing of a fistulous opening in the stomach, that

he uttered his famous dictum: "From this operation to the resection of a piece of carcinomatous stomach there is still only a bold step to be taken." But so far, in all of the operations on dogs, the autopsies had shown that a small piece of the cardiac end of the stomach had been left. It was not until 1894 that Carvallo and Panchon reported a case of total extirpation of the stomach in a cat, which lived for six months. This cat exhibited interesting physiologic phenomena after gastrectomy, of which the increasing indifference to food was the most pronounced. If forced feeding was employed, it appeared able to digest the food, but it persistently refused to eat spontaneously. A point of some interest should be noted, namely, that the stomach of the cat, for anatomic reasons, lends itself much more readily to complete excision than that of the dog.

Experiments similar to those of Gussenbauer and von Winiwarter and of Kaiser were reported by Monari and Filippi, who succeeded in successfully removing practically the entire stomach in two dogs.

In 1884, Phineus Conner of Cincinnati, was the first to perform a complete resection of the stomach in man. This patient, moribund at the time of operation, did not survive the shock and expired on the table.

In 1897, thirteen years later, Schlatter performed a successful total gastrectomy in man, and demonstrated for the first time not only that a human being could live without a stomach but that an anastomosis could be made between the esophagus and the jejunum. This patient, a woman, aged 56, survived one year and fifty-three days, at the end of which time she died of a recurrence of the carcinoma.

This now famous case gave rise to a great deal of discussion from which originated the differentiation between total and subtotal gastrectomy. One year following Schlatter's operation, MacDonald, an American, performed a complete gastrectomy in a man, aged 38. How long this patient survived is not known, as the report merely stated that he left the hospital on the thirteenth day and did not suffer any complication except diarrhea. In the same year, 1898, another American surgeon, Brigham, performed a successful total gastrectomy in a woman, aged 66, and demonstrated that it was possible to reestablish the continuity of the alimentary canal by performing an anastomosis between the esophagus and the duodenum. Brigham was also the first to study the effect of total removal of the stomach on the red blood cells, and pointed out that hematopoiesis was unaffected. Brigham's patient survived more than two years. The fourth successful total gastrectomy was also performed in the United States by Harvie, in 1900, an anastomosis being made between the esophagus and the duodenum.

Then a number of cases were reported in rapid succession until finally the procedure was given a definite place in surgery.

Considerable space in the literature on gastrectomy has been given to a discussion as to just what constitutes a total gastrectomy. This discussion arose at a meeting of the Deutsche Gesellschaft für Chirurgie in 1898, after the report of Schlatter's case. Langenbuch, in 1894, and Schuchardt, in 1895, having previously reported extensive resections of the stomach in which as much as seven eighths of the organ had been removed, were inclined to describe these operations as total gastrectomies. At that time, however, Krönlein defined total resections of the stomach as only those in which both the pylorus and the cardia had been removed, and in which on examination of the specimen one should find a portion of esophagus at one end and of duodenum at the other. Billroth defined a total resection as the suturing of the pylorus to the cardia after removal of the body of the stomach. The definition of Krönlein is obviously more exact. In this study, an analysis has been made of all the cases of total resection of the stomach, 122 in all, of which we have been able to find any record in the literature to date.

We have considered as total gastrectomies only those operations in which the entire stomach has been removed and following which the esophagus has been anastomosed to the duodenum or jejunum. A report on the microscopic appearance of the esophageal or duodenal ends of the specimen removed has been available in only a few instances. Practically, however, from the technical standpoint this is of little importance, for we have recently observed in cadavers that mucosa of the gastric type can frequently be demonstrated in the walls of the esophagus, well above the cardiac sphincter. While, therefore, one can be reasonably certain where the esophagus ends macroscopically and also of the location of the cardiac sphincter, still, microscopically, it is not always possible to be certain where the mucosa of the stomach begins. Failure, therefore, to demonstrate esophageal stratified epithelium in the specimen removed should not constitute an objection to the classification of such a case as total gastrectomy, especially if the excision had been performed above the cardia and distal to the pylorus. In order, however, to be classified as a complete gastrectomy, the cardiac and pyloric sphincters must be removed. This definition has formed our criterion for the classification of total gastrectomies. We have defined as "subtotal" those cases in which any portion of the stomach was allowed to remain at either the cardiac or the pyloric ends. This small portion has arbitrarily been limited to not more than 3 cm. If more than this has been left, we have excluded all such cases from our series.

Because of the lack of sufficient data, we have excluded from our table a number of cases included by other authors which really appear to be cases of gastrectomy, but which, for the sake of accuracy, it was thought best to exclude. In addition to our five cases, we find

reported in the literature sixty-two undoubted cases of total gastrectomy, and fifty-five cases in which less than 3 cm. of stomach had been left. Of the sixty-seven patients in whom total gastrectomies were performed, thirty-six died, constituting a mortality rate of 53.8 per cent, while thirty-one, or 46.2 per cent, recovered. The greatest length of life following total gastrectomy was four years and eight months, in a patient operated on by Zikoff in 1911. The second longest was four years. After that the longevity falls below two years and six months. The records of all the cases were not complete, however, owing to the lack of subsequent reports, so that some of the patients on whom the operation was successful may have lived longer than those previously mentioned. In the few patients in whom follow-up observations have been made, the great majority have succumbed to a recurrence of the carcinoma.

CAUSES OF DEATH

An analysis of the causes of death in the fatal cases is as follows: of the thirty-six fatal cases, twenty-one patients, or 58 per cent of the total number who died, died of peritonitis; eight, or 22.2 per cent, died of shock, and one each died of intestinal obstruction—due to contraction of the mesocolon on the loop of jejunum—stomatitis and enteritis, duodenal fistula, esophageal fistula, hemorrhage, paralytic stroke and cardiac failure. A further analysis of the different methods of repair of the alimentary canal following total removal of the stomach revealed the following facts: in twenty-six patients, the esophagus was anastomosed to the jejunum, and of these, 58 per cent recovered while 42 per cent died. In the second group, in which the esophagus was united to the duodenum, were thirty patients; of these only 47 per cent recovered and 53 per cent died. In the third group, in which anastomosis was not done, were nine cases, all of which, of course, were fatal. Nearly all of these patients were in such a state of shock after the removal of the stomach that they succumbed before the operator had time to perform an anastomosis. In a few, the esophagus was closed off and either the duodenum or jejunum brought out and attached to the surface of the skin. All of these patients died of peritonitis. There were two cases in which information as to the operative procedure was not given. Owing to the fact that the mortality appeared to be greater in the cases in which the esophagus had been anastomosed to the duodenum, a comparative analysis was made between the two groups of cases of total gastrectomy—the one in which the jejunum had been united with the esophagus and the other in which it was united with the duodenum. In the first group of twenty-six esophagojejunostomies, twelve patients died—six of generalized peritonitis, two of shock, one each of ileus, paralytic stroke, blowing out of the duodenal stump followed by duodenal fistula and

stomato-enteritis. In the group of thirty patients in whom esophagoduodenostomy was performed, fifteen died. Of these, ten deaths were due to peritonitis, two patients died of shock, two of causes concerning which there was not sufficiently definite information in the report to determine the cause of death, and one died of cardiac failure. Thus, 67 per cent of the patients in whom esophagoduodenostomy was performed died of peritonitis, in comparison with 50 per cent of those in whom esophagojejunostomy was done. It is difficult to offer a satisfactory explanation of this observation. Several factors may, and probably do, play an important rôle. 1. The duodenum may have been improperly mobilized and under too great tension. 2. The duodenum was possibly more thin-walled and more difficult to suture properly than was the jejunum. The data, however, which we have obtained from the reports of these fatal cases were insufficient to permit any definite conclusion, except to say, on the whole, the use of the jejunum for the reestablishment of the alimentary canal appeared to be a safer procedure than the duodenum.

Subtotal gastrectomies, and by this term we mean the cases in which a small amount of stomach, less than 3 cm., was left at the pyloric or cardiac end or both, were performed on fifty-five patients, forty-one, or 75 per cent, of whom recovered, and fourteen, or 25 per cent of whom died. In comparison with the group in which strictly total gastrectomy was done, this shows a reduction in the mortality of 28.8 per cent. This reduction may be attributed largely to the fact that a small piece of stomach was left, which made the operation easier and technically more safe than when an attempt was made to suture the esophagus directly to the small intestine. Of these patients, 75 per cent recovered, and the length of life following operation was greater than after the total gastrectomies. The greatest length of life after subtotal gastrectomy was twenty-five years, in a patient who was operated on by Peugniez in 1899, and who died of lobar pneumonia in 1924, at the age of 77. Examination of the stomach, which was removed at the time of operation, proved the condition to be carcinoma, and autopsy twenty-four years later revealed the retroperitoneal lymph glands and the liver both involved with carcinoma. The general longevity in this group, although the data in the reports are rather meager, is definitely increased, while the immediate and remote results seem to be correspondingly better. It was noted, however, in some cases, especially those showing leather-bottle stomach, linitis plastica, that it was impossible to do a subtotal gastrectomy, since the entire stomach was involved. In such cases, one was forced to remove the stomach in its entirety. Fortunately, however, for the surgeon, in these cases there seems to be an elongation of the subdiaphragmatic portion of the esophagus, so that further mobilization of it is not usually necessary. Thus, in spite

of the facts which seem to favor subtotal gastrectomy as the operation of choice, it is evident that there will be found occasional cases which will demand a total gastrectomy.

More than 46 per cent of the patients in our series have recovered. It must be borne in mind, too, that at the time of operation these patients are physically in a marked state of malnutrition, so that they are not the best operative risks.

The postoperative course in this group of gastrectomized patients was most interesting and surprising in many ways. First, in all the cases there was no complaint of hunger pains, nor of any sensation of emptiness or fulness in the region of the stomach. Vomiting following the operation did not occur in our cases, and in only six of the cases reported was it troublesome enough to warrant any special notice. In the case of Reid and also in one of ours there was a pronounced and obstinate stomatitis. Reid's patient recovered from the stomatitis and lived more than a year. In our case, the stomatitis seemed to be the factor which resulted in an esophagitis and enteritis, causing the death of the patient twenty-four days after the operation. The enteritis in this case was of a severe type, the stools being voluminous and made up almost entirely of large masses of mucus, in which there were numerous flecks of blood. Kocher had a similar case. Undoubtedly, in these two cases, the primary cause of death was enteritis, as in either case the anastomosis had healed completely. These patients did not seem to be able to absorb food taken by mouth satisfactorily, partly, at least, because of the marked degree of hyperperistalsis which invariably followed its ingestion. Why these patients should develop stomato-enteritis was not apparent. It was our assumption that the general lack of resistance and the lowered state of nutrition were mainly responsible. The cultures from the stools in our case showed only the colon bacillus.

When we studied the postoperative histories as given in the various case reports, one fact stood out in a striking manner, namely, the wide variation in the kind of food administered and the manner of its administration. The apparent differences in the results were so slight as almost to lead one to the conclusion that the character of food and the method of administration were inconsequential. Obviously, however, the diet with the smallest bulk and the highest nutritive value would be indicated, in view of the restricted reservoir space left after the removal of the stomach. The use of the Levine duodenal tube, which is swallowed before the operation and passed through the new stoma into place in the duodenum or jejunum, as the case may be, at the time of the operation and left in situ for several days for immediate feeding, has proved a life saving procedure in our hands, not only in this class of cases but in all forms of gastro-intestinal anastomoses.

Investigation into the effect of resection of the stomach on the digestive processes in man and animals had its beginning in 1882, when Ludwig and Ogata began their study of the famous Czerny dog. The digestion of the dog whose stomach had been almost completely removed seemed to be in no way inferior to that of any other dog. The animal appeared to be perfectly healthy. Autopsy showed that a small portion of the cardia had been left. In 1896, Monari and Filippi did not report any changes in the metabolism in gastrectomized dogs, but the lowest portion of the esophagus and the first portion of the duodenum at the line of anastomosis were dilated and hypertrophied. Not until 1897, however, when Schlatter reported his famous case of gastrectomy were any studies made on the effect of total and subtotal gastrectomy on the human being. In 1898, Wroblewski reported concerning Schlatter's case: (1) The patient had lived for some time without a stomach; (2) small amounts of conjugated sulphates were present in the urine, proving that putrefactive processes in the intestine were not disturbed; (3) the urine showed high acidity; (4) there was no change in the indol, skatol or bile acids in the feces; (5) the vomitus showed combined acids, no free hydrochloric acid, large amounts of lactic acids, and biliary pigment; bile acids and trypsin also were present. In this case, however, it was found subsequently at autopsy that some mucosa of the cardiac portion of the stomach remained. In 1899, Theodore Kocher reported a case of total resection of the stomach, in which the patient died at the end of five days following a protracted and persistent diarrhea. At autopsy, little was found to account for death, except a slight necrosis of the mucosa of the ileum. He attributed the metabolic upset resulting in the diarrhea to necrosis following injury to the superior mesenteric artery, which he vaguely assumed to be due to stretching of the artery during the operation. He stated that anemic necrosis of the mucous membrane may result in diarrhea, and he therefore gave up the use of intestinal clamps.

In 1899, Deganello reported his observations in Tricomi's case of subtotal excision of the stomach. In short, his conclusions, based on observations during the first forty days after operation are: (1) that the nitrogenous substances are not assimilated in the normal manner, and (2) that there are muscle fibers in the feces. He concluded that the stomach is not indispensable to life.

In 1902, Albert Frouin found that excision of the spleen, with and without resection of the stomach, did not have any effect on the digestive processes, and that removal of the spleen did not modify intestinal digestion. The probability that lactic acid originates from the food and acts as a stimulant to the pancreatic ferments in the absence of hydrochloric acid was suggested by Gley in 1902, this observation having been made clinically on several occasions.

TABLE 1.—*Data on Total Gastrectomies*

No. Operator	Date	Country	Result	Complications	Operation	Lesion	Sex	Age	Comment
1. Conner.....	1884	United States	Death from shock by one operation completed	0	Total gastrectomy; no anastomosis done	Carcinoma	F	50	Linitis plastica of stomach, mobile, contracted and pulled down esophagus to greater length than normal, thus leaving considerable portion of esophagus intraperitoneal for anastomosis
2. Langenbuch.....	1894	Berlin, Germany	Well; few weeks	0	Seven eighths of stomach removed; really sleeve resection; end to end, cardia to pylorus	Carcinoma	F	58	
3. Schuchardt.....	1895	Germany	Well; 2½ years	0	5 cm. of stomach remained above excised portion	Carcinoma	M	58	New stomach pouch formed almost as large as original stomach removed; duodenum dilated and formed almost half of this new stomach; no recurrence in new stomach; metastases in lungs
4. Schlatter.....	1897	Switzerland	Well; 1 year and 53 days	0	Total; incision across stomach on a slant or bias, leaving small amount of stomach mucosa posterior which grew over anastomosis like mucosa over an ulcer; esophagus to jejunum	Carcinoma	F	56	Recurrence in abdominal lymph glands; none in dilated portion of esophagus and jejunum
5. Breitung.....	1897	Germany	Well; 1 year	0	Extensive sleeve resection	Carcinoma	F	50	
6. Baldy.....	1898	America	Dead; 36 hours	Hemorrhage shock and infection	No anastomosis attempted; removal; inoperable tumor, probably sarcoma	.....	M	..	
7. Kolaczek.....	1897	Germany	Well; 4 months	0	Seven-eighths resected; gastroduodenostomy	Carcinoma	F	49	
8. Rehn.....	1898	Germany	Well; 1½ years	0	Some cardiac end remaining; gastrojejunostomy	Carcinoma	F	..	
9. Richardson, M. H...	1898	America	Well; 9 months	Fistula from suture line	Seven-eighths resected; gastroduodenostomy	Infiltrating carcinoma	F	53	Recurrence in abdominal lymphatics and mesentery of intestine; none at suture line
10. MacDonald.....	1898	America	Well; 2½ months	Shock	Total resection; Murphy button esophagus to duodenuum	Carcinoma	M	38	

11. Bernays.....	1898	America	Dead; 30½ hours	Shock and infection	Esophagus to pylorus; left some stomach at pylorus, 3 cm.	Carcinoma	M	42	For the first time, intravenous salt solution was given; putting duodenal tube past suture line in stomach; sutured esophagus and intestine before cutting off stomach
12. Brigham.....	1898	America	Well; 2 years	0	Total; esophagoduodenostomy	Carcinoma	F	66	Normal blood formula unaffected
13. Noble.....	1898	America	Died on table	Shock	Total; esophagoduodenostomy could have been done	Carcinoma	M	46	Operation was only begun, not completed
14. Chavasse.....	1899	England	Dead	? Infection; peritonitis	Total resection of stomach; duodenum sewed to skin; esophagus closed	Carcinoma	M	64	No anastomosis between esophagus and intestine
15. Urban.....	1899	Germany	Well; 4 months	0	Small portion of fundus left; subtotal; gastrojejunostomy	Carcinoma	F	35	
16. Riliena y Sans.....	1899	Spain	Well; 4 months	0	Subtotal; esophagopylorostomy; 4 or 5 cm. above pylorus left	Carcinoma	F	40	
17. Körte.....	1899	Germany	Well; 5 months	0	Subtotal; gastrojejunostomy; entero-enterostomy below	Carcinoma	M	42	
18. Tricomi.....	1899	Italy	Well; 4 months	0	Subtotal; gastrojejunostomy; Murphy button; jejunojejunostomy	Carcinoma	F	48	
19. Lambotte.....	1899	France	Dead; 14 hours	Shock and peritonitis	Total; esophagoduodenostomy	Carcinoma	F	64	The stomach in all cases of linitis plastica can be delivered out of abdomen due to lack of perigastric adhesions and also to the great lengthening of the esophagus which seems to result from prolonged traction in these cases
20. Ricard	1899	France	Well; 50 days	0	Subtotal; small part of fundus left; gastrojejunostomy	Carcinoma	F	..	Portion of pancreas removed also
21. Poirier.....	1899	France	Well; 19 days	0	Subtotal; gastrojejunostomy	Carcinoma	M	36	
22. Kocher, T.	1899	Switzerland	Dead; 4 days	Peritonitis; enteritis	Esophagoduodenostomy; total resection	Scirrhus carcinoma linitis plastica	F	44	Diarrhea began on second day; progressively more severe; partial resection of transverse colon
23. von Leersum.....	1899	Holland	Well; 8 months	0	Subtotal; (1) gastroplasty, left some cardia; (2) gastroduodenostomy	Carcinoma	F	22	Diarrhea at first few days

TABLE 1.—*Data on Total Gastrectomies—Continued*

No.	Operator	Date	Country	Result	Complications	Operation	Lesion	Sex	Age	Comment
24.	Tuffier	1899	France	Lived 5 months	0	Subtotal; extensive sleeve resection; pylorus to cardia	Carcinoma	..	?	
25.	Harvie, J. B.	1900	America	Well; 7 months	0	Total; esophagus to duodenum	Carcinoma	F	46	Drawing sensation in epigastrium afterward
26.	Vieira de Carvalho	1900	Brazil	Well; dates not given	0	Subtotal; gastroduodenostomy; some cardia left; Billroth I	Carcinoma	F	46	
27.	Lauwers, Em.	1900	France	Well; 8 months	0	Subtotal; gastrojejunostomy; cardia left	Carcinoma	F	65	
28.	Gallet, A.	1900	France	Well; 2 months	0	Subtotal; no details	Carcinoma	F	59	
29.	Thorne, W. S.	1900	America	Dead; 4 hours	Shock	Total; Murphy button; esophagoduodenostomy	Carcinoma	F	52	
30.	Fralick	1901	America	Dead; 13 days	Cardiac failure or embolus	Total; esophagoduodenostomy	Carcinoma	M	58	
31.	Syme	1902	England	Well; 1 month	0	Subtotal; lateral gastroduodenostomy	Carcinoma	F	55	
32.	Garcia, D. S.	1901	Spain	Well; 1 month	0	Subtotal; gastroduodenostomy	Carcinoma	F	41	
33.	O'Hara	1901	Australia	Well; 6 months	0	Subtotal; duodenum to portion of cardia left remaining	Carcinoma	M	50	
34.	Krause	1901	Germany	Well; 3 weeks	0	Subtotal; 3 cm. at cardia; Billroth I	Carcinoma	F	42	
35.	von Bardeleben	1901	Germany	Well; 2 weeks	0	Total; esophagojejunostomy; calcified bone bobbin employed in suture	Carcinoma	F	52	
36.	Moreau	1904	France	Dead	? Peritonitis	Billroth I; subtotal; 6 cm. cardia remaining	Carcinoma	F	34	
37.	von Dollinger	1902	Austria	Well; 8 months	0	Polya; small amount of cardia remaining	Carcinoma	M	37	
38.	von Herczel	1902	Germany	Well; 11 months	..	Total; esophagojejunostomy	Carcinoma	F	42	
39.	von Herczel	1902	Germany	Well; 11 months	..	Subtotal; Billroth II; Murphy button	Carcinoma	F	43	
40.	Vander Veer	1902	America	Dead	Peritonitis	Total; esophagoduodenostomy	Carcinoma	F	42	

41. Vander Veer.....	1902	America	Lived, 9 months	..	Subtotal; large sleeve resection	Carcinoma	M	35	
42. Recasens.....	1902	Spain	Dead; 15 hours	Shock	Total; duodenum to esophagus	Carcinoma	F	38	
43. Griffith.....	1902	America	Dead; 19 days	0	Subtotal; duodenum to cardia; cardia remaining	Carcinoma	M	..	Died suddenly at home after eating a large meal of cabbage and bacon
44. Robson, Mayo.....	1902	England	Well; 1 year	0	Subtotal; duodenum to cardia; bone bobbin employed in suture	Carcinoma	M	38	
45. Thomas, J. L.....	1902	England?	Well; 1 year	0	Subtotal; three-fourths of stomach removed; gastrojejunostomy	Carcinoma	M	..	
46. Fedoroff, S. P.....	1903	Russia	Dead; 48 hours	Peritonitis	Total; duodenum to esophagus	Carcinoma	M	35	
47. Anderson, Winslow..	1903	America	Dead after 7 weeks	0	Subtotal; duodenum to stomach; Murphy button	Carcinoma	F	37	Probable obstruction at point of anastomosis where Murphy button was employed; patient died of starvation following vomiting
48. Moynihan, B. G.....	1903	England	Dead; 36 hours	Shock	Total esophagojejunostomy	Carcinoma	F	49	
49. Baeckel, Jules.....	1903	France	Dead after 1 year	..	Subtotal; gastroduodenostomy	Carcinoma	F	38	
50. Segâle, J. B.....	1903	France	Well; 2 years	..	Subtotal; duodenum to narrow strip of cardia	Carcinoma	F	56	
51. Ulmann.....	1903	Germany	Well; 1 month	..	Subtotal; no details	Carcinoma	F	62	
52. Gayet and Patel.....	1904	France	Well	..	Subtotal; Billroth II	.....	F	44	Jahanlay operated, using his button for anastomosis
53. Kelling, George.....	1905	Germany	Dead	Peritonitis	Total; esophagus to jejunum; Murphy button	Carcinoma	M	48	
54. Kelling.....	1905	Germany	Dead	Peritonitis	Total; Murphy button; duodenum to esophagus	Carcinoma	M	60	Gives brief account of two other cases in both of which the patient died in three or six days of peritonitis
55. Kelling.....	1905	Germany	Dead	Peritonitis	Total; esophagus to jejunum	Carcinoma	F	48	
56. Ito, H. and Asahara	1905	Japan	Dead	Shock	Total; closed esophagus; duodenal fistula	Carcinoma	F	46	
57. Ito, H. and Asahara	1905	Japan	Well for 6 months	0	Subtotal; little cardia left; duodenum to cardia	Carcinoma	M	46	Carcinomatosis resulted
58. Sironi, Torquato....	1905	Italy	Well; 5 months	Shock	Subtotal; cardia to jejunum	Carcinoma	F	47	Operation in two stages on successive days; first stage, removal of stomach; second stage, anastomosis performed

TABLE 1.—*Data on Total Gastrectomies—Continued*

No.	Operator	Date	Country	Result	Complications	Operation	Lesion	Sex	Age	Comment
59.	Schopf, Franz.....	1906	Germany	Dead; 6 days	Peritonitis	Total; jejunum to esophagus; Murphy button	Carcinoma	F	39	
60.	Leriche, Rene.....	1906	France	Dead; 48 hours	Shock and peritonitis	Total; esophagus and duodenum sutured	Carcinoma	F	48	Peugniez operated
61.	Gregoire.....	1906	France	Well; 7 months	0	Subtotal; anterior gastro-enterostomy	Carcinoma	M	48	Reported by Leriche
62.	Kausch.....	1906	Germany	Dead	Peritonitis	Total; Murphy button esophagus to duodenum	Carcinoma	?	..	
63.	Moynihan.....	1907	England	Well; 3 years 8 months	0	Total; esophagus to duodenum; esophagus sutured	Carcinoma	M	43	Died of symptoms of anemia; no metastases; no recurrence of cancer in the abdomen
64.	Riese.....	1908	Germany	Dead; 5 days	Cardia	Total; esophagus sutured	Carcinoma	F	73	No metastases or infection; suture line excellent
65.	Riese.....	1908	Germany	Well; 6 months	0	Total; esophagus to duodenum	Carcinoma	F	39	Metastases
66.	Soubeyran.....	1908	France	Dead; 24 hours	Shock	Subtotal; anterior gastro-enterostomy	.....	F	61	
67.	Delagenière.....	1908	France	Dead; 12 hours	Shock	Total; esophagus to jejunum	Carcinoma	F	..	
68.	Viannay, Chas.....	1909	France	Dead; 10 days	Pneumonia	Subtotal; gastrojejunostomy following high pylorectomy	Carcinoma	F	41	Another subtotal operation incompletely reported
69.	Goldschwend, F.	1909	Germany	Dead; 2 weeks	Duodenal fistula following drainage	Esophagus to jejunum, end-to-end; total	Carcinoma	F	51	Drainage responsible; Brenner operated
70.	Keding, Paul.....	1909	Germany	Dead; 24 hours	Shock	Total; esophagus to duodenum; suture	Carcinoma	M	66	Müller's cases
71.	Keding, Paul.....	1909	Germany	Dead; 6 days	Peritonitis	Total; esophagus to duodenum closed	Carcinoma	F	43	Müller's cases
72.	Thévenard.....	1910	France	Dead; 14 months	0	Subtotal; little cardia left; no discussion of anastomosis	Carcinoma	M	63	Metastases
73.	von Haberer.....	1910	Austria	Well; 1 month	0	Subtotal; gastrojejunostomy following pylorectomy	Carcinoma	F	49	

74. Häberlin.....	1910	Switzerland	Dead; 16 days	Peritonitis	Total; end of esophagus closed; jejunostomy done	Carcinoma	M	54	
75. Trinkler.....	1911	Germany	Dead; 6 days	Starvation	Total; anterior portion of anastomosis of duodenum and esophagus left open	Carcinoma	F	57	
76. Zikoff.....	1911	Russia	Well; 4 years	0	Total; duodenum to esophagus	Carcinoma	F	56	
77. Arcoleo, E.....	1911	Italy	Well; 5 months	0	Total; lateral, duodenum to esophagus	Carcinoma	F	64	May have left small piece of carcia
78. Burk (Hofmeister)..	1911	Germany	Well; 2 years	0	Total; esophagus into loop of jejunum	Carcinoma	?	41	
79. Schoemaker.....	1912	Germany	Well; 1 month	0	Total; esophagus to duodenum	Carcinoma	M	52	
80. Altschul, W.....	1913	Poland	Dead; 2 days	Peritonitis	Total; esophagus cut off and slipped up; drain drawn down to it; no anastomosis	Carcinoma	No sex	57	
81. Schloffer.....	1913	Germany	Well; 3 years	0	Gastrectomy ?; no data as to operation	Carcinoma	No data		
82. Unger.....	1913	Germany	Well; 6 months	0	Total; esophagus to duodenum	Carcinoma	F	42	
83. Moullin.....	1914	England	Well; 5 years and 1 week	0	Subtotal; esophagus to jejunum	Carcinoma	F	31	
84. von Haberer.....	1917	Austria	Well; 1 year and 3 months	0	Total; esophagus to jejunum	Carcinoma	M	49	
85. Wrede.....	1919	? Germany	Well; no date of operation	0	Total; esophagus to jejunum	Carcinoma	M	36	
86. Schloffer.....	1917	Germany	Well; 8 weeks	0	Total; esophagus to jejunum; Branus anastomosis of afferent and efferent shank	Carcinoma	F	48	
87. Leuret.....	1919	France	Well; 2 months	0	Subtotal; Billroth II	Carcinoma	M	?	
88. Salva Mercade.....	1919	France	Well; no date of operation	0	Subtotal; gastrojejunostomy	Carcinoma	F	55	
89. Sundberg.....	1920	Sweden	Dead; 7 days	0	Total; esophagojejunostomy; anastomosis	Carcinoma	F	65	Paralytic stroke; no peritonitis
90. Drevermann, Paul...	1920	Germany	Well; 2 months	0	Total; esophagus to jejunum	Carcinoma	M	46	
91. Mayo, W. J.....	1921	America	Well; 2 years 11 months	0	Total; esophagus to jejunum	Carcinoma	M	58	Differential blood formula disturbed; blood simulated that of pernicious anemia

TABLE 1.—*Data on Total Gastrectomies—Continued*

No.	Operator	Date	Country	Result	Complications	Operation	Lesion	Sex	Age	Comment
92.	Vander Elst.....	1922	France	Well; 7 months	0	Subtotal; little cardia left; duodenum to cardia	Carcinoma	M	53	
93.	Brun (Kobelt).....	1922	Switzerland	Well; 4 years	0	Total; esophagus to duodenum	Carcinoma	F	45	
94.	Brun (Kobelt).....	1907	Switzerland	Well; 1½ years	0	Total; Branus anastomosis; esophagus to jejunum	Carcinoma	F	?	
95.	Brun (Kobelt).....	1912	Switzerland	Dead; 6 days	Ileus	Total; esophagus to jejunum; Branus anastomosis	Carcinoma	F	?	Obstruction at slit in mesocolon
96.	Brun (Kobelt).....	1912	Switzerland	Well; 2 days	Shock and infection	Total; esophagus to jejunum; Branus anastomosis	Carcinoma	M	?	Necrosis of end of esophagus; transverse colon and spleen removed
97.	Kreuter, Erwin.....	1922	Germany	Dead; 3 days	Shock; infection ?	Total; entero-anastomosis, lower esophago-jejunostomy	Carcinoma	F	40	Probably peritonitis; author describes incarceration of a loop of jejunum; distention of gall-bladder and strip of duodenum
98.	Charrier, A.....	1922	France	Well; 4 years	0	Subtotal; slight amount of cardia left; gastro-jejunostomy, then jejuno-jejunostomy	.....	F	49	Just a small portion of cardia remaining—"cocks comb"
99.	Heuer, G. J.....	1925	America	Dead; 36 hours	Peritonitis	Subtotal; little pylorus left; esophagoduodenostomy	Carcinoma	M	56	No leak in suture line; practically a total; no metastases
100.	Peugniez.....1899 and	1924	France	Well; 3 months	0	? Total; left a seromuscular cuff at end of esophagus; resected all mucosa of stomach; duodenum to esophagus	Carcinoma	M	70	As far as mucosa took all of stomach; Goepel procedure
101.	Kelling....	1923	Germany	Well; 14 months	0	Total; esophagojejunostomy, then jejuno-jejunostomy	Carcinoma	M	51	Death from abdominal recurrence
102.	Kelling...	1923	Germany	Well; 6½ months	0	Total; esophagojejunostomy, then jejuno-jejunostomy	Carcinoma	M	55	Death from abdominal recurrence
103.	Rienhoff.....	1924	America	Dead; 18 days	Enteritis	Total; esophagojejunostomy	Carcinoma	F	64	Patient surgically well; uncontrollable diarrhea with tremendous bloody mucus stools; no peritonitis or leakage of suture line
104.	Schuppel.....	1924	Germany	Well; 4 weeks	0	Total; esophagojejunostomy and entero-anastomosis	Carcinoma	M	44	

105: Hinz.....	1924	Germany	Dead; 24 days	0	Total; esophagoduodenostomy	Carcinoma	F	41	Fistula along drainage tract; died following hemorrhage
106. Hinz..	1924	Germany	Dead	Shock	Total; esophagoduodenostomy	Carcinoma	M	59	
107. Hinz.....	1924	Germany	Well; 1 month	0	Total; esophagoduodenostomy	Carcinoma	F	56	
108. Hinz.....	1924	Germany	Well; 1 month	0	Total; esophagoduodenostomy	Carcinoma	F	48	
109. Peugniez.....1899 and	1924	France	Well; 24 years	0	Subtotal; small amount of cardia remaining	Carcinoma	M	77	Patient lived 24 years after operation; abdominal recurrence in retroperitoneal glands
110. Charrier and Charbonnel	1924	France	Died; 30 days	Peritonitis	Total; no anastomosis made due to shock	Carcinoma	F	51	
111. Pauchet.....	1900	France	Well; 17 days	0	? Total; probably slight amount of cardia left; cardia to duodenum	Carcinoma	F	50	
112. Pauchet.....	1907	France	Dead; 5 days	Pneumonia	Subtotal; gastrojejunostomy following pylorectomy	Carcinoma	M	56	
113. Pauchet or Girrard..	1899	France	Well; 10 months	0	Total; duodenum to esophagus	Carcinoma	?	?	Case not reported except in Pauchet's Surgery
114. Finney.....	1925	America	Well; 1 month	0	Subtotal; stomach to esophagus; some pylorus left	Carcinoma	F	54	
115. Finney...	1925	America	Dead; 6 days	Peritonitis	Subtotal; gastroduodenostomy	Carcinoma	M	71	Leak in suture line
116. Finney.....	1925	America	Dead; 4 days	Peritonitis	Total; duodenum to esophagus	Carcinoma	M	57	
117. Mayo, W. J.....	1920	America	Dead; 5 days	Peritonitis	Total; esophagus to jejunum	Carcinoma	M	36	
118. Mayo, W. J.....	1925	America	Dead; 2 days	Hemorrhage	Total; esophagus to duodenum	Carcinoma	M	41	
119. Reid.....	1925	America	Well; 1½ years	0	Total; esophagus to jejunum	Carcinoma	F	51	Stomatitis; died of metastases
120. Rienhoff.....	1924	America	Well; 4 years	0	Subtotal; pylorectomy with gastrojejunostomy	Sarcoma	M	34	1 cm. stomach remained
121. Brander.....	1926	England	Dead; 10 days	Cardiac failure	Subtotal; pylorectomy with gastrojejunostomy	Sarcoma	M	27	
122. Butler.....	1927	England	Well; 13 months +	0	Gastrojejunostomy; anticolic	Chronic ulcer	M	42	

TABLE 2.—*Data on Gastrectomies*

Total number of cases	122
Number of cases reported in literature	116
Personal communications (Finney, 3; Heuer, 1; Rienhoff, 2; Mayo, W. J., 2)	8
Undoubted cases of total gastrectomy	67
Deaths	36
Recoveries	31
Mortality for total gastrectomy	53.9%
Recoveries following total gastrectomy	46.2%

(By total gastrectomy is here meant complete excision of the stomach, cutting through the esophagus and duodenum)

Greatest length of life following total gastrectomy ..... 4 yr. 8 mo.

It is to be noted, however, that the duration of life herein computed is based on the interval of time elapsing between the date of operation and the date on which the report of the case was published. It is probable that the actual length of life in these cases was in reality much longer

Second longest ..... 4 years

After that longevity falls below ..... 2 yr. 6 mo.

Causes of death

Peritonitis	18 cases	50%
Shock	8 cases	22.2%
Intestinal obstruction	1 case	2.7%
Hemorrhage	1 case	2.7%
Enteritis	2 cases	5.5%
Esophageal fistula	1 case	2.7%
Duodenal fistula	1 case	2.7%
Cardiac failure	2 cases	5.5%
No data	2 cases	5.5%

Subtotal gastrectomy (by this term is meant cases in which a small amount, 3 cm. or less, of the stomach is left remaining at the pylorus or cardia)

Total number of cases	55	
Recoveries	41	75%
Deaths	14	25%

This shows the great importance of leaving even the slightest amount of stomach for purpose of suture. Technically, it facilitates matters very much and decreases the danger of the operation by at least two thirds. The length of life following subtotal gastrectomy is greater than following total gastrectomy; the greatest longevity is in a patient operated on by Pingniez in 1899 who died of pneumonia in 1924. This would suggest that leaving a piece of stomach is by far the safest procedure.

TABLE 3.—*Types of Anastomosis**

1. Esophagus to jejunum	25	58% well	42% died
2. Esophagus to duodenum	30	47% well	50% died
3. No anastomosis	9		100% died
4. No information	2	100% well	
Total	67		

1. Esophagojejunostomy	12
2. Esophagoduodenostomy	15
3. No anastomosis	9
Total number of deaths	36

1. Esophagojejunostomy ..... 12 patients died— 6, or 50%, died of peritonitis
   2, or 16.8%, died of shock
   1, or 8.3%, died of mesocolic ileus, paralytic stroke, blowing out duodenal stump, stomatoenteritis

2. Esophagoduodenostomy ..... 15 patients died—10, or 67%, died of peritonitis
   2, or 13%, died of shock
   2, or 13%, died of no information
   1, or 7%, died of cardiac failure

3. No anastomosis ..... 9 patients died—100%
   45% died of shock
   55% died of peritonitis

* It would seem that a justifiable conclusion to draw from the table is that an anastomosis between the esophagus and the jejunum is to be preferred on the whole to an anastomosis between the esopagus and the duodenum. Further, it should be noted that the operation should be carried through in one stage. In those patients in whom anastomosis was not performed at the primary operation all succumbed, either by shock, peritonitis, or both.

In 1907, Moynihan reported a total gastrectomy in a woman, which was complete according to Kronlein's definition. The patient lived three years and seven months. The autopsy showed nothing but a profound anemia.

The first well controlled experimental work was that done by Carrel, Meyer and Levine. They concluded that in gastrectomized animals there is first a diminished amount of pancreatic and intestinal secretions. This is probably due to the fact that the stimulation from the normal gastric juice is lacking. Proteins in the intestines are therefore at first not fully digested and are absorbed as such. The nitrogen retention is somewhat high; later, however, digestive ferments are restored to their normal power and amount, the nitrogen retention being reduced to normal. In 1912, Adamo reported his study of the subtotal gastrectomy done by Arcoleo, and he demonstrated satisfactorily that there was no disturbance in the metabolism of fat, carbohydrates and protein. He brought out an interesting point—that the esophagus at certain places, especially in its lower third, repeats the structure of the gastric mucosa. He also showed that in a majority of the cases of so-called gastrectomy, all the gastric mucosa had not been removed, and that the secretion from this remaining portion would be sufficient to maintain a beneficient stimulation of intestinal secretion. In those cases in which the stomach had been removed, the lower extremity of the esophagus has, to a certain extent, assumed the function of the lacking gastric mucosa. In 1913, Dagaew, reported that he had not found digestive disturbances in his gastrectomized dog.

In 1920, Drevermann, recapitulated; he pointed out that the dilatation in the lower end of the esophagus and the upper portion of the duodenum, acting mechanically, makes up for the absence of the stomach as a reservoir and that the musculature of the duodenum and of the upper loop of jejunum probably assumes the rôle of motor function of the stomach to some extent. The action of the pepsin-hydrochloric acid digestion is taken over by the trypsin, and the laboratory animals as well as the patients have been well nourished and have enjoyed fairly good health. Examinations of the urine and feces revealed nothing of importance that would indicate any marked physiologic change. Clinically, the cases of atrophy of the gastric mucosa bear witness to the fact that man can digest and assimilate or resorb his food without the aid of gastric secretion, that is, so far as the gastric secretion has to do with hydrochloric acid.

CONCLUSION

In a review of the literature on the function of digestion in gastrectomized animals and patients, we think it can be concluded that patients as well as animals, when properly fed, as regards consistence, amount and periods of feeding, can get along without a stomach. Further, as

far as the pepsin-hydrochloric acid digestion is concerned, it has been shown that physiologic digestion of protein, fats and carbohydrates is not seriously affected by the exclusion of these two elements of the gastric juice. It is extremely difficult, however, for one to be absolutely certain at the operating table, whether or not he has completely removed all trace of the gastric mucosa. It is a matter of microscopic study to say just where the gastric mucosa ends and the esophageal mucosa begins, or vice versa. There is apparently a great deal of individual variation. If the slightest trace of gastric mucosa remains following operation, or, as suggested by Dagaew, if gastric mucosa is present in the lower end of the esophagus, this will probably insure the reestablishment of the function of the gastric mucosa.

That a more beneficial result clinically, physiologically and mechanically is to be expected if one leaves a portion, ever so small, of the gastric mucosa, must be conceded. But, from the present indications, it does not appear to be absolutely necessary for the life or well being of the patient.

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THIRTY-SEVENTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY*

PHILIP D. WILSON, M.D.
LLOYD T. BROWN, M.D.
M. N. SMITH-PETERSEN, M.D.
RALPH GHORMLEY, M.D.
MURRAY S. DANFORTH, M.D.
EDWIN F. CAVE, M.D.
JOHN G. KUHNS, M.D.
BOSTON
C. HERMANN BUCHOLZ, M.D.
HALLE, GERMANY
GEORGE PERKINS, F.R.C.S.
LONDON, ENGLAND
AND
ARTHUR VAN DESSEL, M.D.
LOUVAIN, BELGIUM

CONGENITAL DEFORMITIES

Cervical Rib.—In a study of 303 cases of cervical rib, Adson [1] said that the roentgenograms showed this occurring on the right side in 70 cases, on the left side in 91 and on both sides in 143; the discovery was accidental in 167 cases. In 100 cases the symptoms were so mild that surgical treatment was not advised. In 77 of these, there was indefinite pain in the neck and shoulder with slight radiation down the arm and hand. In 12 cases, pain was exaggerated by rotation of the head or by elevation of the chin. There was a slight atrophy over the ulnar distribution in 12 cases. There was evidence of circulatory disturbance in 4 cases.

Operation was performed in 41 cases. The anterior approach with tenotomy of the scalenus anticus is the preferred operation. It is easier and gives the same result as transcervical approach and resection of the rib without subsequent numbness in the arm, and there is no danger of brachial palsy.

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* This Report of Progress is based on a review of 203 articles selected from 409 titles dealing with orthopedic surgery appearing in medical literature between Feb. 25, 1928, and June 9, 1928. Only those papers that seem to represent progress have been selected for note and comment.

1. Adson, A. W.: Atlantic M. J. **31**:222 (Jan.) 1922.

GROWTH DISTURBANCES

Legg-Perthes' and Köhler's Disease of Tarsal Scaphoid.—Ely,[2] in a report on six cases of Köhler's disease, pointed out that in some of the cases roentgenograms showed changes in both feet while symptoms occurred only in one foot. The diagnosis is based entirely on the roentgenogram; the cause is unknown, and the treatment is symptomatic.

Seldowitz and Zintbaum,[3] studying one case in a boy 6 years of age, concluded that it is a manifestation of rickets. The condition of the blood chemistry and the prompt improvement following antirachitic treatment support this contention.

Katzenstein [4] suggested the transplantation of bone marrow from the tibia in cases of Köhler's disease and Perthes' disease. He claimed that the underlying pathologic process is a lack of regeneration in necrotic bone marrow and thus implants new bone marrow.

Durham and Outland [5] have made a study of the blood serum phosphorus and blood serum calcium in six cases of Perthes' disease. From their observations they reported:

1. Perthes' disease is characterized by a diminution of the serum calcium or phosphorus or of both.
2. The pathology is very similar to that of rickets and osteomalacia.
3. These three conditions may be related etiologically.

[ED. NOTE.—The suggestion that these conditions may be rachitic is interesting, but it would seem a most unusual type. Conditions other than rickets may show blood changes and many bone diseases improve on antirachitic measures. The authors must produce more cases to substantiate their claim. Katzenstein's remedy is radical and would have to be backed by definite evidence of real clinical improvement.]

SCOLIOSIS

Moffat [6] discussed the various operative methods used in the treatment for scoliosis, stated their disadvantages and reported a method which he used in eleven cases.

The technique consists of reflecting a flap composed of periosteum and interspinous ligaments, as in the Hibbs operation, from the spinuous processes and laminae on the concave side. The spinous process at each pole of the incision is cleft and the intervening spinuous processes broken down. A straight three-layer

2. Ely, L. W.: Köhler's Disease, Arch. Surg. **16**:560 (Feb.) 1928.

3. Seldowitz, Morton, and Zintbaum, Leo: Köhler's Disease of the Tarsal Scaphoid Bone: Report of Case, J. A. M. A. **90**:1617 (May 19) 1928.

4. Katzenstein, M.: Zentrabl. Chir. **55**:1872, 1928.

5. Durham, H. A., and Outland, T. A.: J. Bone & Joint Surg. **10**:301 (April) 1928.

6. Moffat, Barclay W.: J. Bone & Joint Surg. **10**:316 (April) 1928.

graft, of sufficient length to reach from one cleft spinuous process to another, is removed from the tibia and inserted as by the Albee technique. The periosteal flap is then sutured around this graft. In this way the graft extends from the beginning to the end of the curve as does a string on a bow. The graft holds the periosteum away from the spine like the ridge-pole of a tent. It also stimulates ossification of the mass of blood and broken bone included by the flap.

Mitchell [7] reviewed 125 cases of scoliosis, 36 of which had been carefully followed. Ten of them were fused. Recumbency in the Hibbs type of plaster or in the turnbuckle jacket of Lovett and Brewster seemed to give the best results in corrective treatment. Fusion is recommended in cases in which progressive deformity is feared. The Hibbs fusion with osteoperiosteal graft was the method of choice.

McKenzie and Starr [8] reported a case of congenital scoliosis with gradual onset of paraplegia, becoming almost complete at the age of 18 years. A decompression was performed at the point of maximum angulation of the spine. The dural sac was found lying tightly against the bone on the inner side of the curve. When the dura was opened, the cord bulged out so that the edges of the dura were separated by at least 1 cm. Within twenty-four hours after the operation there was marked return of muscle function, and nine months later the only positive sign was exaggerated knee reflexes, but no clonus.

[ED. NOTE.—We doubt if Moffat's method of installing a "bow string" graft has any advantage over the osteoperiosteal graft method of fusion which he deprecates. Furthermore, the "ridge pole action" of Moffat's graft violates sound principles of surgical technic. The case of McKenzie and Starr is of considerable interest and importance. Several such cases have been seen by the editors. Changes in the peripheral nerve and cord may be found often in case of scoliosis. The confusion with syringomyelia is easy and must always be cleared up.]

ANATOMIC INVESTIGATIONS

Epiphyses.—Hellman [9] studied roentgenologically the hands of sixty girls from 10 to 14 years of age. Measurements were carefully made of the diaphysis and the epiphysis of the various bones. He divided the process of ossification into five phases: (*a*) distinct wide separation of the shaft and epiphysis; (*b*) narrowing of the epiphyseal cartilage; (*c*) extension of a budlike process of ossification into the cartilage from both ends; (*d*) fusion of epiphysis and shaft—a radio-opaque stage, and (*e*) appearance of trabeculations in the epiphysis. These phases do

7. Mitchell, J. I.: Etiology and Treatment of Scoliosis, Arch. Surg. **16**:680 (March) 1928.

8. McKenzie, K. G., and Starr, C. L.: Arch. Surg. **15**:222 (Aug.) 1927.

9. Hellman, Milo: Am. J. Phys. Anthrop. **11**:223, 1928.

not always follow each other in orderly sequence. Different stages may be seen in different phalanges of the same hand. The first metacarpal ossifies like a phalanx and raises the question whether this may not be a phalanx of the thumb rather than a metacarpal bone. The greatest growth in the bones of the hand occurs during the thirteenth year. This preceded the greatest increase in height by one year. Ossification in the epiphyses of the hand first appeared from twelve years and thirty-six days to twelve years and eleven months. Ossification was complete from fourteen years and three months to fifteen years. The entire process lasts less than three years, although a single phase may last a year or more. Ossification is first completed in the distal phalanges. The most active growth of bone occurs during phases *b* and *c*. The most prolific growth occurs at the diaphyseal end of the cartilage.

Todd and D'Errico [10] showed that the sternal epiphysis of the clavicle does not always completely ossify. Marked excavation of the sternal end, often erroneously called posterior dislocation, has no influence on the character of the sternal epiphysis except to modify its shape. The clavicle is a true limb bone forming in cartilage with epiphyses at both ends. The sternal epiphysis begins to unite from about the twenty-first to the twenty-second year, union not being completed until the twenty-fifth year. An explanation cannot be given for this delay. The lateral epiphysis of the clavicle unites in the twentieth year and usually ossifies. There is no significant difference in the sexes or between the white and black races.

Rogers [11] demonstrated that "an epiphysis for the styloid process of the fifth metarsal bone occurs between the ages of 11 and 17 in 28 per cent of subjects" (fifty cases examined).

Bipartite Carpal Scaphoid.—D. M. Faulkner [12] reported a case of bilateral bipartite carpal scaphoid. From his study of the condition, he believed that bipartite carpal scaphoid, with cartilaginous surfaces between the two fragments, does occur, and secondly, that this abnormal joint may be the seat of an arthritis.

In his case an arthritic condition was present, on account of which the scaphoids were removed and an opportunity for study of the anatomic changes afforded.

[ED. NOTE.—This is an important observation because of the ease with which one might erroneously diagnose such a condition as a fractured scaphoid.]

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10. Todd, T. W., and D'Errico, J., Jr.: Am. J. Anat. **41**:25 (March 15) 1928.
11. Rogers, Lambert: J. Bone & Joint Surg. **10**:197 (April) 1928.
12. Faulkner, Donald M.: J. Bone & Joint Surg. **10**:284 (April) 1928.

Range of Motion at Hip Joint in Men.—Moore and Vaughn,[13] from a study of the ranges of motion in the hip joints, gave the following summary:

1. The total range of motion, in flexion and extension, at the hip joint in normal young adult males is approximately 146 degrees on both sides, the average flexion being 114 degrees and the average extension 32 degrees.

2. The range of motion in the individual lies between 121 degrees and 175 degrees.

3. Deviations from the average range are slightly more marked in extension.

4. The flexion-extension ratio decreases as the total range increases.

5. The range of motion shows no relation to height or weight except when the extremes are reached.

6. No marked tendency to right or left-"Leggedness" is demonstrable from the range of motion that exists.

BACK STRAIN AND FOOT STRAIN

Billington[14] sent out a questionnaire to 250 American orthopedic surgeons. From seventy-nine replies received, he summarized the following:

The causes of low backache are broadly, arthritis, trauma and posture. These three types, though apparently clearly differentiated, are in reality closely associated and one form may induce one or both of the others.

Differentiation between sacro-iliac and lumbo-sacral lesions causes the greatest difficulty. This is important if operative procedure is to be undertaken.

In acute traumatic conditions, the treatment is rest followed by physiotherapy. In severe cases, rest in bed or in plaster jackets or shells with or without traction, is necessary. In some cases of acute sacro-iliac strain manipulation may be used. In chronic backache, all postural defects must be corrected and when arthritic or toxic irritation is present, all foci of infection must be removed, elimination promoted and the diet regulated. Sacro-iliac supports and spinal braces and corsets may be used, together with postural exercises, muscle training and physiotherapy.

In old chronic and persistent cases, some ankylosing operation may give relief, provided the diagnosis has been carefully made. The removal of large transverse processes does not seem satisfactory.

[ED. NOTE.—Such a summary as this is worth while, only in that it shows how difficult it is to generalize on the treatment for back strain and its allied conditions. It seems to us that each case demands a course of treatment of its own. Generalization has kept down progress in the proper direction. Specification will get us much farther.]

Hypermobility of the First Metatarsal Bone.—Morton,[15] in studying the foot, found that "hypermobility of the first metatarsal bone" is due

13. Moore, J. B., and Vaughn, J. O.: J. Bone & Joint Surg. **10**:248 (April) 1928.

14. Billington, R. W.: J. Bone & Joint Surg. **10**:290 (April) 1928.

15. Morton, Dudley J.: J. Bone & Joint Surg. **10**:187 (April) 1928.

to unusually free motion in the joint between the inner cuneiform and scaphoid bones, and between the inner and middle cuneiforms.

Analysis of a large group of cases of metatarsalgia, in which hypermobility or brevity of the first metatarsal was present (singly or in combination), indicates that the disorder is essentially a physiologic one, comprising an improper distribution of stresses borne by the different metatarsal bones; it is not attended with any measurable change in the relative position of the metatarsal heads as inferred by the term "depressed anterior arches." This term is therefore regarded as inapt, and as conducive to misapprehension of the real nature of static disorders in this part of the foot.

TUMORS

Metastases to Bone in Primary Carcinoma of Lung.—Hirsch and Ryerson [16] reported four cases of primary carcinoma of the lungs with metastases to the bones. Careful postmortem examination was performed in all cases, all parts of the body being included. Two of these cases were diagnosed during life as "primary endothelioma." The diagnosis was based on specimens removed at operation. The authors pointed out that reports of cases of so-called endotheliomas of bone are frequently based on tissue removed at operation and not on postmortem examination with a careful study of the lungs. In metastatic carcinoma of the bones, and in "primary endothelioma," the lungs should always be included among the probable sources.

Multiple Myeloma.—Geschickter and Copeland [17] reviewed 425 reported cases and added 13 cases. The incidence is twice as common in men and may begin between the ages of 35 and 70 years. The disease is characterized by pain, insidious at onset and increasing in severity, multiple tumor formation, deformities of the chest, spine and long bones; spontaneous multiple fractures; gastro-intestinal symptoms, cough, neurologic disturbances and symptoms of nephritis.

Bence-Jones bodies were found in only 65 per cent of the cases. Anemia was present in many, and in 70 of 100 cases the leukocyte count was normal with a tendency to mononucleosis. The roentgenogram shows multiple punched-out areas, usually in the ribs, skull, spine and long bones. Section of the tumor tissue shows little stroma with small round cells of the plasma or lymphoid type. The prognosis as to life is rarely over two years. Treatment is symptomatic.

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16. Hirsch, E. F., and Ryerson, E. W.: Metastases of Bone in Primary Carcinoma of the Lung: Review of So-Called Endotheliomas of Bones, Arch. Surg. **16**:1 (Jan.) 1928.

17. Geschickter, C. F., and Copeland, M. M.: Multiple Myeloma, Arch. Surg. **16**:807 (April) 1928.

CIRCULATORY DISTURBANCES

Thrombo-Angiitis Obliterans.—Allen and Brown [18] have studied 200 cases of thrombo-angiitis obliterans. Pathologic studies were made on thirty amputated limbs and eight resected veins. The pathologic changes were as described by Buerger, except in two respects; i. e., there was rarely evidence of acute inflammation, and internal proliferation was found out of proportion to the age of the patient. Although organisms were not obtained from the vessels on bacteriologic study, the authors thought that the disease was of infectious origin.

Silbert and Samuels [19] reported their results in the use of the Pachon oscillometer in thrombo-angiitis obliterans. Cases were studied with the oscillometer, readings being taken at intervals of from two to three months. Examination with the oscillometer frequently revealed pulsation when none can be felt on palpation. When oscillometric examination showed pulsation at the ankle, the prognosis for relief of symptoms is good, even if ulceration or actual gangrene is present. If the examination with the oscillometer gives a zero reading, the prognosis for saving the extremity is poor, the process being more advanced. In this group if gangrene was already present, the outlook was poor.

TUBERCULOSIS

Drainage of Abscesses in Spinal Tuberculosis.—Steindler [20] said that symptomless abscesses should not be interfered with. The most dangerous extension of abscesses is backward into the spinal canal. This may cause paraplegia. In lower dorsal and lumbar abscesses, there may be extension under the fascia of the iliopsoas or retroperitoneal extension. Evacuation of retromediastinal abscesses is indicated when they embarrass the function of the adjacent organs. This is best done by the costotransversectomy of Menard. Occasionally the operation will relieve paraplegia where laminectomy fails. A trocar may be used if the location of the abscess is certain. For retroperitoneal abscesses, drainage is indicated only when the abscesses are large or infected. This should be done by a posterior incision along the erector spinae or lateral border of the quadratus lumborum.

[ED. NOTE.—This is one of the most important problems met in tuberculosis of the spine. As an emergency measure, drainage by incision is certainly indicated. As a therapeutic measure, drainage would help greatly, but for the danger of establishing sinuses which is so real as to contraindicate it.]

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18. Allen, E. V., and Brown, G. E.: Ann. Int. Med. **1**:535 (Feb.) 1928.

19. Silbert, Samuel, and Samuels, Saul: Thrombo-Angiitis Obliterans: Prognostic Value of Oscillometer, J. A. M. A. **90**:831 (March 17) 1928.

20. Steindler, A.: J. Kansas Med. Soc. **28**:37 (Feb.) 1928.

Type of Tubercle Bacillus.—Cobbett [21] reported two cases of tuberculosis of the joint in adults, in which large numbers of tubercle bacilli of the human type were present in the pus. He reviewed the evidence on the relative frequency of the bovine and human type in tuberculosis of bones and joints. In table 1, he quotes the results of A. S. Griffith (of Cambridge, England), who has studied this matter for twenty years.

Morrison [22] injected into guinea-pigs material from 100 patients whose condition was diagnosed clinically as tuberculosis. He found that in almost a third of the cases, inoculation into guinea-pigs failed to produce this condition. The material injected was almost always caseous debris or pus. Patients with a positive Wassermann reaction were not included in the series. Morrison did not consider the failure to

TABLE 1.—*Results of Study by Griffith*

	Number of Cases	Percentage of Human Type
All ages	541	81.3
Under 10 years	327	74.6
Under 5 years	102	70.6
English and Scottish cases compared:		
Scottish cases: all ages	28	71.0
English cases: all ages	389	81.7
Scottish cases: under 10	22	63.6
English cases: under 10	288	75.4

TABLE 2.—*Data Compiled by Morrison*

	Bovine Percentage	Human Percentage
Lymphatic gland infection		
Children	84	16
Adults	38	62
Bone infection		
Children	30	70
Adults	18	82

produce tuberculosis in the animal due to a wrong diagnosis. Table 2 contains data regarding the relative frequency of the bovine and human bacillus.

In the two types, the results of treatment were different. After a period of two years all the patients with bovine infection were in good health, and their lesions quiescent; whereas more than 18 per cent of the patients with human infection were dead.

[ED. NOTE.—These articles, coming from two different sources and presenting figures so much in agreement, are of much interest. Failure to produce tuberculosis in the guinea-pig by inoculation does not necessarily rule out this condition. Much depends on the method of inoculation.]

21. Cobbett, Louis: Brit. M. J. **1**:626 (April 14) 1928.
22. Morrison, John T.: Lancet **22**:319 (Aug. 18) 1928.

Tuberculosis of the Tarsus.—Pouzet [23] analyzed the localization of tuberculosis in the tarsus. It showed a special predilection for the scaphoid, or more particularly, the "inner portion" of the tarsus. He distinguished: (1) tuberculosis of the scaphoid, occasionally with an abscess, often resembling Köhler's disease; (2) tuberculosis of the scaphoid-cuneiform joint, and (3) tuberculosis of the astragaloscaphoid joint.

Of twenty localizations in the mediotarsal region, sixteen were astragaloscaphoid. Of twelve localizations in the anterior tarsal joint, eleven were in the scaphoid-cuneiform joint. In sixteen cases, treatment by immobilization in thirteen resulted in cures in eight cases and an arrested condition in five cases. Seven operations were performed in cases in which the condition had been arrested and three in cases in which there had not been previous treatment by immobilization. The author noted: (1) the frequency of recurrence following conservative treatment, and (2) the occurrence of static difficulties after healing.

Tuberculosis of the Wrist.—Sorrel and Longuet [24] have reviewed twenty cases of tuberculosis of the wrist in children. In only two of these had the process extended to all the bones. None of the children was under 4 years of age. They expressed the belief that this was due to the fact that up to the age of 4, the bones of the wrist are almost entirely cartilaginous, and between the ages of 4 and 12 they are so well surrounded with avascular cartilage that progression of the disease is stopped by the cartilage. Conservative measures of treatment, rest, heliotherapy and splinting resulted in excellent functional results in 80 per cent of the cases.

PYOGENIC INFECTIONS

Osteomyelitis.—Platt [25] stated that in children the primary focus was in the metaphysis; diaphyseal osteomyelitis, in which the initial focus developed toward the middle of the shaft was uncommon. In the adult, the initial focus often remained latent.

Platt warmly supported the observations and practice of the Toronto school. He contended that in all but the fulminating cases there existed a prodromal phase, lasting two or three days, during which the diagnosis could be made on the basis of: (1) fixed pain at the end of a bone, and (2) localized metaphyseal tenderness. Simple drainage of the metaphysis during this stage sufficed to cure the disease before the infection had spread, and prevented bone necrosis. Platt urged that

23. Pouzet, F.: Rev. d'orthop. **15**:308 (July) 1928.
24. Sorrel, E., and Longuet, J.: Rev. d'orthop., Jan., 1928, vol. 15.
25. Platt, Harry: Proc. Roy. Soc. Med., 1927-1928, p. 1378.

efforts should be made to diagnose the disease in this prodromal phase. Osteomyelitis was most often confused with acute monarticular rheumatism, which, in his opinion, was a rare condition. In all doubtful cases, exploratory puncture of the bone should be done.

The early diagnosis of acute osteomyelitis in the flat bones was difficult and the prognosis bad. In diagnosing osteomyelitis of the pelvis, three signs were of value: (1) referred pain radiating down the thigh on the same side, (2) pelvic tenderness on rectal examination and (3) a sense of deep resistance between the trochanter and the iliac crest.

The principle of local treatment was to establish early and effective drainage, without infecting healthy tissue and without further devascularizing infected and uninfected bone. Three types of drainage were practiced:

1. Metaphyseal drainage, according to the technic of Starr. This was indicated if the disease was diagnosed in the prodromal stage.

2. Diaphyseal drainage—guttering the shaft of the bone. This was indicated when the medullary canal was widely infected, which occurred only late in the disease.

3. Subperiosteal resection of the diaphysis. This had no justification except in the neglected patient in whom the whole shaft was riddled with infection. There would always be a risk of nonregeneration, owing to destruction of the periosteal tube from the acute inflammation. With early diagnosis, metaphyseal drainage should become the routine and only treatment.

Martens [26] said that he regarded articular rheumatism, contusion and typhoid as particularly common differential problems. He advocated primary opening of the bone marrow before a periosteal abscess has formed. Plenz, in discussing this paper, regarded the general condition as of more importance in indicating operation than the local condition.

Jura [27] carried out certain experimental work on the transmission of osteomyelitis from rabbit to rabbit from which he stated the following conclusions:

> It is proven that from the culture of certain germs (*Staphylococcus aureus* and *albus*, Bacillus of Eberth) isolated from typical cases of osteomyelitis, it has been possible by means of appropriate technique (plasmolysis, trituration, the action of certain bacteriolytic substances and successive filtration through Berkefeld W. or Chamberland L 3) to obtain filtrates, capable of reproducing the osteomyelitic lesions in rabbits, either when introduced into the vein or when introduced into the medullary cavity.

26. Martens, A. O.: Zentralbl. f. Chir. **55**:1617, 1928.
27. Jura, Vincenzo: Policlinico **35**:77 (Feb. 15) 1928.

Lesions have been reproduced in series of rabbits by inoculating them by the same methods with emulsion of bone marrow infected with osteomyelitis produced by inoculation of the filtrates.

The filtrable osteomyelitic virus can be cultivated in anaerobic cultures. It has a notable resistance to heat and to certain chemical agents (chloroform); it is destroyed by alkalis and by acids; it is endowed with certain antigenic properties as brought out in testing for complement deviation, testing with serums of patients with osteomyelitis or with rabbits treated with the virus.

Patients affected with osteomyelitis show a specific cutaneous hypersensibility toward it, revealed especially by an immediate or almost immediate reaction to intradermal inoculation.

These facts, therefore (he believed), proved what he had already concluded that osteomyelitis is associated with a filtrable virus.

[ED. NOTE.—The first two articles are in line with all modern trends in the treatment for acute osteomyelitis. The third paper brings up a question that can be supported only by confirmation of the observations by others and must overcome a host of bacteriologic evidence to the contrary.]

INFANTILE PARALYSIS

Aycock,[28] in studying the distribution of cases of poliomyelitis, found a greater number of cases in the younger age groups in urban than in rural communities. The same was found to be true in the distribution of diphtheria and measles. This he regarded as significant in that the mode of distribution for measles, diphtheria and poliomyelitis is probably the same. The virus of poliomyelitis is believed to attain a distribution equal to that of measles and diphtheria, in the course of which the majority of persons become immunized either subclinically or through unrecognized attacks of the disease.

CHRONIC ARTHRITIS

Report of the German Orthopedic Congress.—At the last meeting of the German Orthopedic Congress, a large amount of attention was devoted to the consideration of chronic arthritis.

Heine,[29] who examined 15,000 joints in 1,000 cadavers at the Schmorls Institute in Dresden, discussed the pathologic changes found in chronic arthritis, particularly in that type which he called genuine or primary arthritis deformans. The disease begins with typical changes affecting the articular cartilage, consisting in swelling, edema, fibrillation and ulcerative degeneration. These changes are of a degenerative character and justify the use of the term degenerative arthritis in designating

28. Aycock, W. L.: Am. J. Hyg. **8**:35 (Jan.) 1928.
29. Heine, J.: Ztschr. f. orth. Chir. **49**:7, 1928.

this type, although the affected joints are still capable of recovery and repair. Repair may be accompanied or followed by deforming reactive processes. Heine was unable to accept Axhausen's theory that the primary change of arthritis deformans, is a necrosis of the articular cartilage. He occasionally found such areas, but there was no evidence to indicate that they were of much importance. He placed emphasis on the point that the resorptive changes so commonly seen in unused parts of certain joints have a striking resemblance to, but have nothing to do with, the primary changes of arthritis deformans, although they may favor the process.

Traumatic lesions of cartilage may heal even in persons of advanced age, but then they often lead to arthritis deformans. The same is true in respect to lesions of cartilage produced by infection or chemical injury, but the resulting arthritic changes have no relationship to true arthritis deformans.

Axhausen has taken up and supported the old teaching of Rokitansky and Ziegler that in many instances arthritis deformans arises from primary changes in the epiphyses, and he has attempted to distinguish a cartilaginous and an osseous form. Heine refuted this theory, stating that such primary osseous changes in the epiphyses may heal, and that when arthritis deformans develops following such osseous necrosis, it always begins with degenerative changes in the cartilage.

He did not consider the hypertrophic spurs so commonly seen in arthritis deformans as altogether specific for this disease, pointing out that they are also seen with resorptive changes and even in normal joints, as for example in the hip joint. These spurs are caused by adaption to the pressure forces at work, and their progress is in reverse relation to the resistance of the cartilage.

The changes found in the synovial membrane are not always related to the epiphyseal changes. The synovial fluid is generally increased and frequently contains coagulated fibrin which may change into bone or cartilage and lead to the formation of loose bodies. Heine did not agree with the frequently stated opinion that the cartilage may show amyloid degeneration, but thought that the red color of the staining reaction indicated the presence of glycogen instead of amyloid. He admitted the fact and importance, however, of fatty degeneration.

He regarded the changes found at the junction of bone and cartilage as of special importance. Pommer taught that even in the early stage arthritis deformans produced characteristic histologic changes that could be recognized. These consisted of the marrow and vascular spaces breaking through the zone of ossification. Heine, on the other hand, did not regard such appearances as specific for arthritis deformans, since he had frequently encountered them in physiologic resorptive processes. The cartilaginous nodules which have been found so fre-

quently within the subchondral marrow spaces were explained by Heine as being in direct connection with, and extensions from, the cartilage.

According to Heine, the order of the involvement of the large joints was as follows: knee, elbow, hip and shoulder. If the small joints were included, the first metatarsophalangeal joint would range right after the knee joint.

Finally, Heine discussed the etiology of arthritis deformans and stated his conclusion that a constitutional disposition must be assumed. Mechanical or functional influences lead only to secondary deforming changes. The theory of constitutional origin is supported strongly by the fact that in patients with carcinoma or tuberculosis, arthritis deformans is relatively rare. In patients with endocarditis, on the other hand, it is rather frequent. The theory of an infectious or vascular origin cannot be accepted. Heine explained that the so-called malum coxae senilis is usually of the secondary type, in consequence of Legg-Calvé-Perthes' disease or of a fracture of the hip.

Speaking of chronic spondylitis, Krebs [30] (Aix-la-Chapelle) stated that it is no longer possible to uphold the idea that a clear distinction can be made between spondylitis deformans and spondylitis ankylopoetica. For the latter condition, he proposed the term spondylitis ulcerosa. In many cases, various combinations of both conditions may be seen. The diagnosis of spondylitis may often be made even in the early stage, if the roentgenograms are carefully studied, as they frequently show distinct but minute changes. He stated that studies made of the blood of many patients with chronic arthritis showed an increase in calcium salts.

Vulpius [31] (Heidelberg) discussed the operative and mechanical treatment for chronic arthritis. In the case of the hip joint he disliked arthrodesis and preferred to obtain a mobile joint, even at the expense of support and weight-bearing. For the knee and shoulder, in severe cases, however, he preferred joints that were solidly ankylosed.

Spitzy [32] (Vienna) stated that in his opinion arthritis deformans is caused by abuse of the joints in conditions of endocrine dysfunction. His observations showed that reduction of body heat in chronic arthritis is always harmful, and he considered heat to be one of the best remedies. Moreover, he thought it important for the patient to keep the affected joints moving as much as possible. Bedridden patients must be made to walk again, first reducing any contractures that may be present. He regarded extra-articular osteotomy as the best method for correcting adduction contracture of the hip. For flexion contracture of the knee he

30. Krebs: Ztschr. f. orth. Chir., 1928, vol. 49.
31. Vulpius: Ztschr. f. orth. Chir., 1928, vol. 49.
32. Spitzy: Ztschr. f. orth. Chir., 1928, vol. 49.

advocated forcible reduction, and he found this method effective in contractures of as much as 120 degrees. He considered the elbow joint well adapted to arthroplasty and the wrist joint for resection.

Schede [33] (Leipzig) expressed himself as in agreement with the views of Payr, who regards arthritis deformans as an aging of the joints. He discussed other phenomena produced by aging of the human body, as for example those which are exhibited by the muscles and which are of importance in arthritis. He stated that the aging of muscles gives rise to the well known ptotic syndrome, consisting of visceroptosis, faulty posture, weak feet, venous stasis of the legs and feet and tender hardenings in the muscles. Aging muscles are less apt for kinetic than static work. Hence the circulatory and respiratory systems suffer; the carbohydrate metabolism within the muscles declines owing to lack of oxygen, and acidosis develops. This affects the joints, as is shown by the acid reaction of the synovial fluid. Moreover, strong muscles are the best protection of the joints, and aging weak muscles cannot give proper support. Schede stated that the aging of muscles may begin to show itself as early as the period of puberty, and that the symptoms should be recognized and active prophylactic measures instituted.

According to Fliegel [34] (Vienna), it is possible to distinguish the following four ankylosing or stiffening conditions of the spine:

1. Spondylitis of Strümpel-Marie or Bechterew's disease, which are practically identical. This disease is characterized by the early involvement of the ligaments and of the joints of the vertebrae. When the disease affects primarily the posterior longitudinal ligament, the spine remains straight; otherwise a progressive kyphotic deformity develops.

2. Primary arthritis deformans. This disease affects the small posterior articulations of the spine but not the ligaments. The small joints of the hands and feet may likewise be involved.

3. Calcinosis intervertebralis. This is a disease of the intervertebral cartilages which leads to their degeneration and final disappearance, generally without the formation of exostoses on the vertebral margins.

4. Spondylitis deformans (hypertrophic spondylitis). Generally, this does not lead to a complete stiffening of the spine.

Hackenbroch [35] (Cologne), discussing arthritis deformans of the first metatarsophalangeal joint, distinguished a primary and secondary form. The latter is usually seen in deformed feet. The primary form is less common and is seen in young persons of the asthenic type, with long, flexible, but otherwise normal feet, who have subjected their feet

33. Schede: Ztschr. f. orth. Chir., 1928, vol. 49.
34. Fliegel: Ztschr. f. orth. Chir., 1928, vol. 49.
35. Hackenbroch: Ztschr. f. orth. Chir., 1928, vol. 49.

to excessive strain by reason of occupation or participation in strenuous sports. Here the stiffening is induced by contraction of the flexor tendons. Often the sesamoid bones are likewise affected. When operative treatment is indicated, Hackenbroch recommended resection of the joint and implantation of fat tissue.

[ED. NOTE.—The editors have before them only the summary of the discussion of chronic arthritis by members of the German Orthopedic Congress, and in consequence must refrain from commenting on the views that were expressed. The symposium evidently covered the subject from a wide variety of viewpoints and even in the abstracted form certain parts of the discussion appear highly suggestive. We regret the lack of a standard terminology for designating the various types of chronic arthritis. This renders a considerable part of any symposium such as this either unintelligible or actually misleading to one who is not familiar with the nomenclature in use. There has recently been organized an International Committee for the Study of Chronic Arthritis with a large representation of interested physicians from each of the countries. It is to be hoped that this committee will attack and solve this problem. If it succeeded in nothing more than setting up a practical and workable classification of the disease which would be universally accepted, its existence would more than be justified.]

Basal Metabolism in Chronic Arthritis.—Swaim and Spear [36] have studied the basal metabolism in 200 patients with chronic arthritis, 94 infectious, 54 atrophic, 52 hypertrophic. They came to the following conclusions: 39 per cent of these patients had an abnormal metabolic rate. The age of the person, duration and activity of the disease apparently did not have a great effect on the metabolism. In the infectious group, the number with a plus rate was slightly greater than the number with a minus rate. In the atrophic, and especially in the hypertrophic, types, the tendency was toward a minus rate. Only 54 per cent in the latter group were normal, and this group showed a greater variation from normal than any of the other groups. As this study showed 39 per cent variation from normal metabolic rates in patients with chronic arthritis, the writers expressed the belief that determinations of metabolic rate should be made in all cases of chronic arthritis. In some patients showing a metabolism above normal, but an erratic and unstable metabolic rate, the metabolism has dropped far below normal after administration of thyroid; as the amount of thyroid administered has been increased, the metabolic rate has gradually risen to normal, and the curve has become more stable. This has been ascribed to relief of nervous tension. It would seem, then, that the use of thyroid in

36. Swaim, L. T., and Spear, L. M.: Boston M. & S. J. **197**:350 (Sept. 1) 1927.

some cases would be of distinct benefit; this has been borne out by the clinical experience.

Streptococcus Infection of the Intestinal Tract.—An infection of the intestinal tract with either *Streptococcus viridans* or *Streptococcus hemolyticus* was found by Keating [37] in more than ninety patients suffering with chronic arthritis whom he studied. On the other hand, on examinations of 100 stools of patients suffering with various other diseases, these organisms were found in only 10 per cent. In one patient affected with gout, positive culture from the stools were not obtained. Keating suggested that the intestinal infection found in chronic arthritis is secondary to infection of the tonsils or air passages. When intestinal infection is present, removal of the primary foci in the teeth and tonsils does not effect a cure.

Arthritis Due to Paratyphoid Bacillus.—Lewis [38] reported the case history of a child, aged 14 months, with a subacute arthritis of the knee. On the fifteenth day after the onset, aspiration was performed on the knee, and a pure culture of paratyphoid B bacillus was grown from the thick, glairy exudate. The child never showed any symptoms of typhoid, nor could paratyphoid organisms be cultured from the stools or urine.

O-Iodoxybenzoic Acid in Infectious Arthritis.—Stein and Taube [39] treated 102 patients who had chronic arthritis with o-oxybenzoic acid in the form of a solution amiodoxyl benzoate (Abbott). The patients were treated in the outpatient department and were allowed to go home in from two to four hours after receiving the injection. Two patients with rheumatic fever were made worse by the drug. Of the 100 patients treated, 31 claimed temporary relief, but almost every one of them returned later with either the same or exaggerated symptoms. No effect was produced on the swelling or joint changes. The uniformly poor results obtained caused the authors to discontinue the use of the drug.

[ED. NOTE.—Although hailed with high hopes and tried with enthusiasm, the experience that has thus far accumulated with o-iodoxybenzoic acid in the treatment of patients with chronic arthritis on the whole has been disappointing. The editors are of the opinion that it may afford symptomatic relief temporarily but that this will not be found to be permanent. Reliance ought not to be placed in it to the exclusion of the general and constitutional methods of treatment that have demonstrated their value in the past.]

(To be Continued)

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37. Keating, P. M.: South. M. J. **21**:263 (April) 1928.

38. Lewis, J. T.: Brit. M. J. **2**:1080 (Dec. 10) 1927.

39. Stein, H. C., and Taube, Norman: O-Iodoxybenzoic Acid in Treatment of Infectious Arthritis: Report of 102 Cases, J. A. M. A. **90**:1608 (May 19) 1928.

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THE SIGNIFICANCE OF CHANGED INTRATHORACIC PRESSURES*

EVARTS A. GRAHAM, M.D.
ST. LOUIS

When reduced to fundamental mechanics, the act of inspiration is a simple one which is accomplished by a sudden enlargement of the thorax and a consequent diminution of pressure within it. Air, therefore, rushes down the trachea so that the pressures on the outside and the inside of the thorax will be more nearly equalized. Similarly, the act of expiration is the reverse of that of inspiration. By muscular action the thorax is diminished in size, the intrathoracic pressure is increased, and air is forced out of the trachea. Changes of intrathoracic pressure, therefore, loom large as factors necessary in respiration and consequently in the maintenance of life.

It is a curious paradox that although the recognition of the importance of maintaining the normal intrathoracic pressure relationships was an effective barrier against the development of surgical procedures in conditions of the chest until recently, now procedures which result in at least a temporary increased thoracic pressure are advocated in the treatment of many pulmonary conditions. These procedures are particularly the use of artificial pneumothorax, thoracoplasty and paralysis of the diaphragm by operations on the phrenic nerve. In other words, the conditions which formerly were regarded as dangerous to change are now regarded as advantageously changeable in the patient who has two of the most common lesions with which the thoracic surgeon has to deal, pulmonary suppuration and pulmonary tuberculosis. One is tempted to go even farther and to say that some apparently regard measures of compressive therapy as almost a panacea for pulmonary lesions.

Generalization is always fraught with danger when dealing with matters of such varied nature and of such varied conditions as clinical problems. The remarkable results which follow properly selected surgical measures for the compression or collapse of a lung in some cases should not blind one to the fact that in other cases death has been caused promptly after, and probably because of, the accomplishment of the desired compression. I have had the experience more than once of seeing a patient die of pulmonary edema after the performance of a thoracoplasty which had seemed technically satisfactory. A similar

*From the Department of Surgery, Washington University Medical School and Barnes Hospital.

experience by others must have been responsible, to some extent at least, for the rather general preference for a two or more stage operation over an operation performed in one stage. It is inconceivable that any procedure which may seriously alter the intrathoracic pressure can have only beneficial effects in the presence of disease and yet be harmful in a normal person. Eleven of twenty-four early deaths after thoracoplasty reported by Stöcklin [1] were said to be due to "heart failure," in which presumably the same sort of pulmonary edema occurred. In the table of results after thoracoplasty for pulmonary tuberculosis, given in Alexander's book [2] on the subject, many other deaths from pulmonary edema are reported. Cardiocirculatory failure is stated by Alexander to be one of the most frequent causes of death after the operation.

It has seemed to me that at this time it might be particularly appropriate to call attention again to some of the effects of altering the normal intrathoracic pressures and to emphasize some of the dangers as well as some of the possible benefits to be derived from such alterations. It seems especially fitting to do so at this time, because since the technic of thoracoplasty is relatively so easy, many surgeons who have no special training in, or rational point of view concerning, scientific thoracic surgery, are performing this operation; in fact, some who have had no extensive training even in the principles of surgery are performing it. It seems to me that this association should take a firm stand against the idea that thoracic surgery is nothing more than the practice of rib cutting. It should also decline to take into its membership those applicants who are only rib cutters, whose only announced claim to membership is the fact that they have performed a certain number of thoracoplasties for this, that and the other condition. This association, on the other hand, should assume a conspicuous leadership in advancing the fundamental knowledge of all those features of disease which pertain to surgical conditions of the thorax. It is for that reason that the association has welcomed in the past, and should continue to welcome, into membership others than surgeons who have been able to make significant contributions to the fundamental knowledge of thoracic disease.

The principle of compressive or collapse therapy has been of the utmost value in properly selected cases of pulmonary tuberculosis and of pulmonary suppuration, but it can probably be made entirely safe

1. Stöcklin, H.: Beitrag zur Behandlung der Lungen-Tuberkulose mit extrapleuraler Thorakoplastik, Beitr. z. Klin. d. Tuberk. **51**:350, 1922.

2. Alexander, J.: Surgery of Pulmonary Tuberculosis, Philadelphia, Lea & Febiger, 1925, p. 310.

and satisfactory only when there is a much greater knowledge of its effects on fundamental physiologic processes. In the normal thorax, any great increase of pressure in one pleural cavity results in pressure disturbances not only on the lung of that side but also on the lung of the other side. A corresponding deviation of the mediastinal structures also occurs. The factors which permit an increase of pressure on one side without deviation of the mediastinal structures and a corresponding effect on the opposite lung are those which accomplish a stabilization or an immobilization of the mediastinal structures. Such an immobilization is effected usually by adhesions or by an induration of the mediastinal pleura.[3] Collapse therapy may, therefore, have serious results if injudiciously employed in cases in which the mediastinum is capable of much movement. Thoracoplasty, because of its permanence, will obviously be more dangerous in such cases than artificial pneumothorax, the effect of which can be counteracted by the withdrawal of air if necessary. Likewise, for similar reasons it would seem advisable to perform the operation of thoracoplasty in more than one stage in spite of the tendency on the part of many to complete the procedure in one stage. Unquestionably, much of the high mortality from empyema during the war was due to the harmful effects of compression and of deviation of the mediastinal structures caused by the creation of an open pneumothorax from too early drainage of a pleural cavity. It will be unnecessary to review the evidence in favor of this opinion here.

Other effects of compression are known, some of which may be beneficial and others harmful. Again some of the effects which may be beneficial under certain conditions may be harmful under others. Sauerbruch[4] long ago found that an increased intrathoracic pressure caused by an open pneumothorax in experimental animals raised the venous pressure in the extremities, a result which seemed to indicate that the flow of venous blood into the heart was impaired. This is an important observation which should be investigated further, particularly with reference to the conditions which underlie the production of pulmonary edema as a result of increasing the intrathoracic pressure too much. Questions also arise in this connection, which have not been satisfactorily answered, concerning the resistance of a normal and of a diseased myocardium respectively to changes of intrathoracic pressure directly on it. Dr. Duff Allen is now engaged in an investigation of

3. For a more extensive discussion of this matter see Graham, E. A.: Alterations of Intrapleural Pressure and their Significance, Medicine **3**:417, 1924.

4. Sauerbruch, F.: Zur Pathologie des offenen Pneumothorax und die Grundlagen meines Verfahrens zu seiner Ausschaltung, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **13**:399, 1904.

these matters in the laboratory at Washington University Medical School. I have observed that patients who have been in bed for too long a time do not tolerate thoracoplasty so well as others and are more likely to develop a pulmonary edema after the operation, a point which suggests that perhaps a myocardium maintained in a more robust condition by some exercise is less likely to be harmfully affected by marked increases in the intrathoracic pressure.

The vital capacity may be markedly reduced by thoracoplasty. This reduction in itself is not necessarily a serious matter, but if the patient should develop pneumonia, cardiac decompensation or some other condition which in turn may lower the vital capacity, or if his vital capacity for any reason should happen to be low at the time of the thoracoplasty, then the operation may have serious consequences. The significance of the vital capacity in thoracic surgery has been discussed by me in several other papers;[5] its general significance in medicine and surgery has been discussed by Yates[6] in an important paper given before this association in 1924. Yates has emphasized particularly the importance of conserving the integrity of the pulmonary circulation, and he has developed the interesting and suggestive hypothesis that the vital capacity is regulated by an air cell-capillary gear of which the weaker part is the circulatory segment.

Knowledge of the effect of increased intrathoracic pressure on the pulmonary blood and lymph circulations is at best fragmentary. This whole question needs much more intensive study than it has received up to the present time. The observations of Cloetta[7] that in slight compression there is more blood in the lung and in marked compression there is less blood, have never been, so far as I know, satisfactorily proved or disproved; Brauer,[8] however, denies that a lung which is only even slightly compressed contains more blood. Rost[9] has concluded that in any case of either compression or relaxation, a lung contains actually less blood than before the compression.

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5. Graham, E. A.: The Importance of the Vital Capacity in Thoracic Surgery, J. A. M. A. **75**:992 (Oct. 9) 1920 (footnote 3).

6. Yates, J. L.: The Significance of Vital Capacity in Intrathoracic Therapy, Arch. Surg. **10**:477 (Jan.) 1925.

7. Cloetta, M.: Eine neue Methodik zur Untersuchung der Lungenzirkulation, Arch. f. exper. Path. u. Pharmakol. **63**:147, 1910; Ueber die Zirkulation in der Lunge und deren Beeinflussung durch über- und unter-druck, ibid. **66**:409, 1911; In welcher Respirationsphase ist die Lunge am besten durchblutet, ibid. **70**:407, 1912.

8. Brauer, L.: Ueber die operative Behandlung der Lungentuberkulose, Deutsche med. Wchnschr. **47**:548, 1921.

9. Rost, F.: Pathological Physiology of Surgical Diseases, Philadelphia, Blakiston's Son & Company, 1923.

Andrus,[10] in a paper before this association in 1924, showed that after the ligation of the main bronchus of one lung of a dog a marked reduction in the amount of blood in the atelectatic lung occurs. The presence of the atelectasis seemed to be the decisive factor in the diminution of the blood supply; a similar result would therefore be found in any case of atelectasis whether produced by compression or otherwise. White and Gammon [11] found experimentally that if fat is injected intravenously after the creation of a unilateral pneumothorax all of it will go to the opposite lung. This observation would indicate strongly that at least with the pressure used by these investigators the capillaries of the affected lung are sufficiently narrowed to prevent the entrance into them of the larger fat droplets. Similarly, Krampf [12] observed that after producing pulmonary embolism with small cubes of paraffin the creation of a pneumothorax repeatedly once a day for from five to seven days causes the area of embolic congestion to be changed to one of infarction. In addition, however, to the scant knowledge of the manner in which the pulmonary circulation of blood is affected by increases in the intrathoracic pressure, one cannot always be certain of how these changes will affect the diseased process.

Knowledge of the effect of increased thoracic pressure on the lymph flow has for the most part also been largely conjectural. The work reported by Dr. Dolley from the laboratory at Washington University is apparently the first conclusive evidence that an increase of intrathoracic pressure definitely impedes the flow of thoracic lymph, although other experiments which suggested this fact had been performed previously.

For example, Shingu [13] of Brauer's clinic, using rabbits and cats, found that after inhalation of soot the production of a unilateral pneumothorax greatly prolonged the time required for the elimination of the material on that side as compared with the other side. He attributed this result to a reduction of the lymph drainage caused by an immobilizing effect of the pneumothorax on respiratory movement. Tendeloo [14] also had concluded, but without any experimental basis, that the amount

10. Andrus, W. C.: Observations on the Cardiorespiratory Physiology Following the Collapse of One Lung by Bronchial Ligation, Arch. Surg. **10**:506 (Jan.) 1925.

11. White, W. C., and Gammon, A. M.: Some New Features of Interest about the Pulmonary Circulation and the Fate Therein of Intravenously Introduced Fats, Tr. National Assn. for Study and Prevention of Tuberculosis, Tenth Annual Meeting, 1914, p. 221.

12. Krampf, F.: Deutsche Ztschr. f. Chir. **189**:216, 1925.

13. Shingu: Beitrag zur Physiologie des kunstlicher Pneumothorax und seiner Wirkung auf die Lungentuberkulose, Beitr. z. klin. Tuberk. **11**:1, 1908.

14. Tendeloo: Studien über die Ursachen der Lungenkrankheiten, Wiesbaden, 1902.

of thoracic lymph drainage is directly proportional to the amount of respiratory activity.

Diminution of the thoracic lymph flow may at times be beneficial, but at other times harmful. Nageli [15] considered that the improvement after thoracoplasty is due to the fact that toxic absorption is reduced by the diminished respiratory motive factor in the lymph flow. Sometimes, however, the interference with lymph flow may be harmful if Noetzel's [16] contention is correct that his experimental finding that the addition of a pneumothorax enabled otherwise innocuous bacteria to induce an empyema was to be explained by a disturbance of the lymph and blood circulations caused by the pneumothorax. Bettman [17] likewise found that the introduction of either air or saline solution into the pleural cavity greatly diminished its capacity to absorb india ink. It is uncertain whether or not the factor of increased pressure caused by the pneumothorax had anything to do with the result, but it is at least interesting that a procedure which is commonly used for compression therapy, namely, pneumothorax, will diminish the absorption of particulate matter from the pleural cavity.

There can be no doubt that under suitable conditions an increase of the intrathoracic pressure will have a beneficial effect, but there can also be no doubt that under other conditions it may be harmful. Future investigations must be relied on to enable one to state more precisely what the conditions are which may make compressive therapy a harmful procedure.

Although knowledge of the effects of compression therapy is unsatisfactory, knowledge of the effects and of the potentialities of decompression therapy is still more meager. This is probably largely due to the fact that there are apparently not so many conditions which lend themselves to the application of decompressive measures. The strikingly beneficial effects which follow the withdrawal of air or of fluid from the pleural cavity in cases of tight pneumothorax or of large accumulations of fluid are so well known as to require no comment. Furthermore, they suggest that in cases of severe dyspnea caused by extensive thoracic tumors a similar beneficial effect might be expected from a decompression induced by the removal of several ribs or by a longitudinal splitting of the sternum. Unfortunately, however, in those cases in which the dyspnea is the greatest the serious symptoms are

15. Nageli, T.: Die Aenderung der serologischen Reaktion des Blutes nach der extrapleuralen Thorakoplastik, Beitr. z. klin. Chir. **90**:351, 1914.

16. Noetzel: Ueber die Infection Bakterien, Resorption der Pleurahohle, Arch. f. klin. Chir. **80**:678, 1908.

17. Bettman: Reaction of Pleura to Particulate Matter Especially in Presence of Pneumothorax, Arch. Surg. **10**:523 (Jan.) 1925.

usually due to the fact that the tumor closely encircles the large vessels and the trachea; therefore, they cannot be relieved merely by a decompression accomplished by the removal of ribs or by the splitting of the sternum. An interesting discussion [18] of this subject occurred in the 1922 meeting of this association, in which cases of unsuccessful attempts to improve the dyspnea of some mediastinal tumors by a longitudinal splitting of the sternum were reported by Yates, Hedblom and myself.

More interesting possibilities of giving relief by thoracic decompression suggest themselves with reference to certain cases of cardiac disease. At this meeting of the Congress at which the subject of heart disease has been designated as the principal topic for discussion it seems particularly appropriate to present this matter, however briefly, to this association for thoracic surgery. In 1902, Brauer,[19] then of Heidelberg but now of Hamburg, now famous also for much pioneer work in thoracic surgery although himself a physician, proposed to Peterson that the third, fourth and fifth left ribs should be removed in a case of chronic mediastinopericarditis in order to permit the heart to contract without the necessity of pulling on the rigid wall of the chest. The operation was carried out with conspicuous success by Peterson, and to it the name of cardiolysis was given by Brauer. The idea, strictly speaking, was not to accomplish decompression, but rather to permit the heart more freedom in action by allowing it to pull against soft yielding tissues instead of against a rigid bony wall.

Despite numerous striking successes, this operation, to my mind, has never received the popularity which is due to it. In one case in which I carried out the typical operation of Brauer five years ago, the patient was at the time completely incapacitated because of cardiac decompensation which was not improved by digitalis. He has now, however, been continuously at work for more than four years in a clerical position at which he works ten hours daily and earns a sufficient income to support himself and wife. In 1912, Summers [20] of Omaha was the first to perform the operation in this country. Dr. Elsworth Smith, who referred the patient to me, has made a careful and exhaustive review of all cases of cardiolysis reported in the literature. His article is as yet unpublished, but he has consented to allow me to quote the results from it. Seventy cases in which the operation has been performed have been reported. The condition in twenty-one of these cases, according to the authors, was refractory to medical treatment before the opera-

18. Discussion on Surgery of the Mediastinum, Arch. Surg. **6**:290 (Jan.) 1923.

19. Brauer, L.: Die Kardiolysis und ihre Indicationen, Arch. f. klin. Chir. **71**:258, 1903.

20. Summers, J. E.: Observations on a Case of Mediastinopericarditis Treated by Cardiolysis (Brauer), J. A. M. Sc. **145**:74, 1913.

tion; these cases are probably after all the best test of the value of the operation. Nevertheless, of the patients in this group of cases, 71 per cent were reported as having been improved by the operation, and of these no less than 24 per cent were rehabilitated sufficiently to return to work. Only 5 per cent of those who survived the operation were not improved. Twenty-four per cent of the twenty-one patients died within three months after the operation. The mortality of 24 per cent may seem to some too high to justify the operation, but it would be hardly fair to ascribe this mortality entirely to the operation, since many of the patients were apparently on the road to death anyway. The other side of the picture, the fact that 24 per cent were returned to work from an otherwise apparently hopeless state of invalidism, seems to me to be the feature which should be emphasized. It is incomprehensible to me why this operation has found so little favor, and I am impelled to feel that the explanation lies in an ignorance on the part of medical men as to the potential benefits in properly selected cases.

As has been already stated, Brauer regarded the chief value of his operation, not in decompression, but in the freedom of action permitted by contractions on a yielding instead of on a nonyielding structure. In 1907, however, Alexander Morison [21] of Edinburgh, in ignorance of Brauer's ideas, proposed "the principle of operative interference to relieve intrathoracic pressure when the mere bulk of the heart was the chief feature in the case." On hearing of the success of the first operations for cardiolysis, he concluded that perhaps the benefit derived from the operation was not entirely to be explained in accordance with the ideas of Brauer, but that perhaps also the factor of actual decompression was important. I shall quote from his article:

> The question arises, whether mere bulk is not a disadvantage in these circumstances, and whether, therefore, the justification does not arise for attempting to relieve pressure by the removal of a portion of the ribs and cartilages in the precordia, in obedience to the indications given by the precordial bulging of children, even when it may be assumed that pericardial tethering is absent.

Acting on his ideas, he persuaded Mr. E. C. Stabb of Edinburgh to carry out an operation similar to cardiolysis on a young man, 19 years of age, whom he had been treating with only moderate success for six years previously for an aortic regurgitation with a greatly enlarged heart. This young man also had very severe precordial pain. The operation was well tolerated, and marked improvement occurred in the patient's condition, with almost a complete disappearance of the distressing pain. A subsequent report [22] a year later stated that the

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21. Morison, A.: On Thoracostomy in Heart Disease, Lancet **2**:7 (July 4) 1908.

22. Morison, A.: Further Report on a Case of Thoracostomy for Heart Disease, Lancet **2**:1494 (Nov. 20) 1909.

patient had continued to show improvement and was able to work steadily as a hat maker.

Since this report, others have occurred from time to time, chiefly in the French literature, describing marked improvement after decompression operations for large hearts. Without wishing in any way to minimize the brilliance of Dr. Elliott Cutler's result in his first case of valvulotomy, I nevertheless have often wondered if some of the result may not be ascribable to a decompression, since in the published report it was stated that the presystolic murmur of the mitral stenosis persisted after the operation and that marked bulging of the split sternum occurred and remained after the operation. Dr. Cutler will doubtless tell more about this matter in his paper.

It is not meant by these remarks in any sense to advocate a wholesale practice of decompression operations for enlarged hearts. The dangers inherent in such an advocacy are too readily apparent. The idea has been rather to suggest that occasionally a case may arise in which a decompression may be helpful in a patient who otherwise would be doomed to helplessness and perhaps to an earlier death. The opinions of others on this matter will be welcome, particularly from those members and guests who are not surgeons. One of the great advantages and opportunities which this association presents is to become a forum for the interchange of opinions and ideas of those who have expert knowledge of thoracic disease. It is for that reason that I particularly invite discussion on these somewhat rambling remarks.

LOBAR PNEUMONIA

CONSIDERED AS PNEUMOCOCCIC LOBAR ATELECTASIS OF THE LUNG

BRONCHOSCOPIC INVESTIGATION *

POL N. CORYLLOS, M.D.
Professor of Clinical Surgery, Cornell University Medical College
AND
GEORGE L. BIRNBAUM, M.D.
Assistant in Surgical Research, Cornell University Medical School
NEW YORK

In a preliminary paper we have given the results of our experimental investigations on lobar pneumonia in dogs, briefly stating the experimental facts on which we have based the theory that lobar pneumonia must be considered as the pneumococcic variety in the large category of obstructive atelectasis of the lung.

In the present paper, we propose to develop this theory further and to show that a new light may be thrown on our knowledge concerning lobar pneumonia which may help to explain in a simple and clear way a great number of its features that have heretofore been inexplicable; furthermore, we shall show that pneumonia, up to now considered an exclusively medical disease, may be as much surgical as medical, and as such require an emergency treatment, necessitating a closer collaboration of the thoracic surgeon, the internist and the bronchoscopist.

We shall first develop our conception of lobar pneumonia and then present the experimental data on which such views are based. We shall then discuss successively the etiology, pathogenesis and pathology, clinical evolution, roentgen observations, diagnosis and state our conclusions.

THEORY

We believe that pneumococcic infection of the bronchial tree by itself is not sufficient to produce lobar pneumonia.[1] Pneumococci may be present in the bronchial tree and give rise to a pneumococcic bron-

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* From the Department of Surgical Research, Cornell University Medical College, the First Medical Division of New York Hospital (Cornell) and the Fourth Medical Division of Bellevue Hospital.

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1. Because of the great predominance of pneumococcus in lobar pneumonia (95 per cent of the cases), we shall consider exclusively pneumococcic pneumonia. What is true for pneumococcic pneumonia is applicable to other organisms which may be concerned in the same syndrome.

chitis without producing "lobar pneumonia," or they may pass through the lung, enter the blood stream and localize in such other parts of the organism as the peritoneum, the meninges or the pleura.

For the production of the acute syndrome, known as "lobar pneumonia," another factor besides the presence of pneumococcus is necessary; this is the occlusion of a bronchus by the pneumococcic bronchial exudate. This marks the onset of the clinical syndrome in which we find united the clinical features of an acute obstructive atelectasis and an acute pneumococcic cellulitis. The topography and size of the bronchus occluded, the duration of the occlusion, the virulence of the microbes and the rapidity of the development of pneumococcic cellulitis will determine the features of lobar pneumonia. Occlusion of a large bronchus with pneumococcus of low virulence will produce a syndrome closer to massive atelectasis than to lobar pneumonia. On the contrary, occlusion of a bronchus with highly virulent pneumococcus will cause a more typical and toxic lobar pneumonia.

In its earliest period, lobar pneumonia presents radiographic and auscultatory symptoms of a lobar atelectasis, namely: a wedge-shaped shadow, more or less marked, absence of respiratory breathing, with a rather tympanitic dulness over the affected area, and often a homolateral displacement of the mediastinum and elevation of the diaphragm on the affected side. Simultaneously, the cellulitis spreads from the hilar portion of the lobe to the periphery, accompanied by the phenomena of pneumococcic inflammation, namely, congestion, interstitial and lymphatic infiltration, vascular engorgement and production of the characteristic fibrinous exudate.

The roentgen and physicial signs of lobar atelectasis, which are due to the obstruction of a central bronchus, appear to proceed from the periphery to the center of the lobe, because the absorption of the alveolar air is more marked in the periphery of the lobe, where the alveoli are more numerous. The symptoms of the pneumococcic cellulitis, on the contrary, spread from the central infectious "plug" to the periphery, following the lymphatic and interstitial tissue, so that after the initial and transitory period of simple atelectasis there is a period of pneumococcic invasion. Engorgement, red hepatization, gray hepatization and resolution follow one another from the center to the periphery. When the hilar portion of the lobe shows engorgement and even red hepatization, in the periphery, we still find alveoli, more or less atelectatic, but free from exudate. When gray hepatization starts in the hilar portion of the lobe, the more peripheral parts show red hepatization; when, after the resolution, the proximal portion of the lobe has drained and become aerated, the peripheral part still contains fibrinous exudate, more or less liquefied by the action of the proteolytic

ferments produced by the white blood cells. Thus the shadow persists, even after the crisis in pneumonia, and gradually disappears from the center to the periphery. This correlates the facts, heretofore inexplicable, that the roentgen precede the clinical signs and persist after their disappearance—features noticed in carefully studied postoperative pneumonia (A. O. Whipple). If this has not been reported so often in medical cases of lobar pneumonia, it has probably been due to the lack of sufficient roentgen data.

The simultaneous development of atelectasis and pneumococcic cellulitis explains the less marked displacement of the mediastinum in pneumonia than in massive atelectasis. Besides, the lesion is often limited to one lobe, and the inflammatory swelling of the interstitial tissue and alveolar exudation prevent shrinkage of the lung to the extent found in simple atelectasis.

The crisis or lysis is due to the disintegration of the fibrinous exudate which characterizes gray hepatization and to the sudden liberation of a large bronchus, thus allowing a sudden drainage of the affected lobe. If this liberation occurs as early as the second, third or fourth day, there is an abortive form of the disease or an early crisis; if it is delayed, there is an unresolved form of the condition. If the liberation is complete and rapid, a crisis follows; if it is gradual, the healing occurs by lysis.

After the crisis, the lobe is rapidly liberated of the pneumococcic exudate; the inflammation subsides as quickly as an acute abscess opened in time; the repair of the lesion is rapid, leaving almost no residuum after a short period. On the other hand, if the obstruction of the bronchus is prolonged, greater alterations of the bronchi and the parenchyma of the lung will follow, thus explaining the unresolved pneumonias and the production of postpneumonic abscess, bronchiectatic lesions, etc.

The localization of the pneumococcic atelectasis is dependent on the same laws as is massive atelectasis. For purely mechanical reasons it is more frequent in the inferior lobes, particularly the right, because of the more vertical direction and larger size of the right bronchus. Thus is explained the easier occurrence of an accumulation in it and the more difficult expulsion of the pneumococcic exudate.

In postoperative lobar pneumonia, this mechanism is even more apparent. This disease starts as lobar atelectasis, in which the relatively sterile occluding mucus is secondarily infected, generally by the saprophytic group IV pneumococcus. This microbe is usually found in the expectoration of these patients, often in pure culture. On the degree of secondary infection of the obstructing mucus and the type and growth of pneumococcus concerned will depend the further evolu-

tion of the disease. If the liberation of the bronchus is early, as is fortunately frequent, the disease is of the atypical form described by A. O. Whipple, which is comparable to the French "maladie de Woillez," the characteristics of which are "its very early appearance after operation, its usual localization in one of the lower lobes, engorgement of the capillaries, lesser air content of the affected lung, and exudate poor in fibrin." The more prolonged the obstruction and the earlier the infection of the occluding mucus with pneumococcus, the more the picture resembles that of pneumonia. So it is that surgeons describe "lobar pneumonia complicating massive atelectasis," whereas internists speak of "atelectasis complicating pneumonia." In post-operative cases, pneumococcus or streptococcus pneumonia, an anaerobic infection, abscess or gangrene of the lung may occur, depending on the virulence and nature of the micro-organisms infecting the mucous "plug."

The only distinction between massive and patchy atelectasis is a difference in the size of the obstructed bronchus. If a large bronchus is occluded, there will be a massive or lobar atelectasis; if several small bronchi are obstructed, a patchy or lobular atelectasis will occur. The same classification is applicable to infectious atelectasis, and it may be said that the relation of lobar pneumonia to lobular or bronchopneumonia is the same as that of massive atelectasis to lobular or patchy atelectasis. The richer in fibrin, and consequently the more viscid, sticky and tenacious the exudate is, the greater are the possibilities for the occlusion of a larger bronchus and the development of lobar pneumonia.

The pneumococcus is the most frequent agent of lobar pneumonia, for it is the organism that calls forth exudate high in fibrin and viscid enough to occlude a large bronchus.

In lobar pneumonia, sudden and massive drainage by the liberation of a large bronchus is more apt to occur, so that crisis is the rule. In bronchopneumonia, liberation of the disseminated small bronchioli occurs slowly and successively, so that healing takes place by lysis, although a crisis may occur when there is simultaneous drainage of a greater number of lobular bronchioles.

There is one factor in the production of the occlusion of a bronchus the importance of which has not been sufficiently investigated. This factor is the ciliated epithelium which lines the bronchi. Its importance in the "breaking up" and the elimination of the column of mucus is probably greater than is generally believed. It may well be that the rapid onset of lobar pneumonia in drunkards exposed to cold or after immersion in water is partly, at least, due to the decrease or inhibition of movement of the cilia. This could explain, to some extent, the often

mentioned "cold reflex" or "decrease of resistance of the lung under the influence of cold." Moreover, permanent lesions of the ciliary apparatus might explain the frequent recurrence of pneumonia often in the same lobe. By the same mechanism, infectious or allergic common "colds" and pharyngotracheobronchitis may prepare the ground for pneumonia by their deleterious effects on the ciliated epithelium.

The foregoing conception of pneumonia readily explains:

(1) The lobar distribution of the lobar pneumonia.
(2) The mechanism of postoperative pneumonia.
(3) The sudden onset of the disease.
(4) The more frequent localization in the inferior lobes, particularly the right.
(5) The atypical, abortive and delayed forms.
(6) The crisis, which cannot be explained by immunologic factors alone.
(7) The often reported homolateral displacement of the heart and elevation of the diaphragm on the affected side, especially in children.
(8) Atelectasis complicating pneumonia or pneumonia complicating atelectasis.
(9) The predisposition to recurrence of pneumonia.
(10) The relation between "colds" and the development of pneumonia.

In favor of the foregoing conception of lobar pneumonia are a great number of experimental and clinical arguments.

EXPERIMENTAL DATA

The following problems were investigated:

(1) The experimental production in dogs of pneumococcic "lobar pneumonia."
(2) The interpretation of the clinical and roentgenographic signs in experimental lobar pneumonia with special reference to massive atelectasis.
(3) The influence of bronchial obstruction on the production and evolution of the disease after infecting the corresponding lobe with a culture of pneumococcus.
(4) The influence of decreased oxygen tension on the development of pneumococcus cultures.
(5) The effects of the liberation of a bronchus by aspiration of the exudate contained in it.

Technic.—The dog is not the ideal animal for this type of investigation because its resistance to pneumococcic infection is so great as to necessitate the use of great numbers of virulent organisms. For this reason, when pneumonia was produced, it was almost constantly fatal. In those cases in which a small amount of virulent culture or larger amounts of older culture were used, the only signs were a rise of temperature, leukocytosis and slight (if any) roentgenographic changes; these symptoms lasted for two or three days without notably affecting the general condition of the animal. Notwithstanding these handicaps, we selected the dog for our work, the reasons for the selection being the great mobility of its mediastinum which offers negligible resistance to displacement of the organs contained in it (such as the heart and trachea), and the large size of its bronchial tree which renders bronchoscopic manipulation easy. The monkey would have been a more suitable animal because of the greater clinical resemblance of experimental pneumonia produced in it to pneumonia in man. Unfortunately, its larynx and tracheobronchial tree are so narrow that accurate bronchoscopic manipulation is almost impossible.

From eighteen to twenty-two hour old broth cultures of a very virulent strain of pneumococcus type I were employed. These were procured from the laboratory of Dr. Russell Cecil in Bellevue Hospital. The virulence of these cultures varied little, and generally 1 cc. of a 1:1,000,000 dilution of culture killed white mice within from twenty-four to seventy-two hours when injected intraperitoneally. The technic used for the production of pneumonia was, in many respects, similar to the method described by Lamar, Meltzer, Wollstein and their associates, differing particularly in the use of the bronchoscope and roentgenography. The animals were first thoroughly examined to rule out accidental pulmonary diseases, then anesthetized by intraperitoneal injection of a 10 per cent alkaline solution of iso-amyl ethyl barbituric acid (amytal). We generally used 55 mg. of this drug per kilogram of weight of the animal. Its use insures an anesthesia lasting several hours and excludes the possibility of respiratory irritation, as may occur in inhalation narcosis.

The Jackson standard bronchoscope (40 cm. by 9 mm.) was employed. Its introduction into dogs weighing from 4 to 12 Kg. is easy, and no traumatism of the trachea or bronchi is produced when it is carefully manipulated.

The pneumococcus culture was atomized in the desired bronchus with the Clerf specimen collector, converted into an atomizer by attaching a small piece of fine rubber tubing to the metallic tube which projects into the glass tube of the instrument. The apparatus was sterilized, and the culture was placed in the glass tube and insufflated into the desired bronchus by connecting the tube for compressed air to the part designed for the attachment of the aspirating tube.

Generally from 5 to 15 cc. of nineteen hour old culture was used. In order to avoid the objection that the occlusion of the bronchus might be due to the amount of injected fluid alone, we centrifugated the same amount of culture and took up the residue in from 0.5 to 1 cc. of sterile broth. It was found that the use of this concentrated culture gave a greater percentage of pneumonia (95 per cent), whereas, with nonconcentrated culture, only 65 per cent of the dogs developed the disease, probably because a portion of the insufflated culture was coughed out of the bronchus.

Before the insufflation of culture, a control roentgenogram was taken, and the temperature, pulse rate and respiration were noted. A few hours after the injection another picture was taken, and subsequently at least one every twenty-hours hours. Blood counts and blood cultures were taken daily.

There is an important detail of roentgen technic in dogs, namely, the strict thoracic symmetry necessary in estimating the displacement of the heart to one side or the other. Even a slight distortion may lead to erroneous interpretation. By a symmetric picture is meant one in which the perpendicular ray passes through the middle of the sternum and the middle of the corresponding vertebrae. The criterion of symmetry is that the line joining the spinal process must coincide with a line drawn through the middle of the vertebral bodies. Because of the "shiplike" form of the dog's chest, even slight rotation of the animal may give greater distortion on the plate. After repeated failures, an apparatus was constructed (figs. 1 and 2) which gave us full satisfaction.

For the obstruction of a bronchus after insufflation of culture, the technic described in detail in our paper on "Massive Atelectasis" was used.

The bacteriologic examinations were carried out with the collaboration of Dr. Torrey and Dr. Kahn of the department of Public Health and Preventive Medicine of Cornell University Medical College.

Experimental Production of Lobar Pneumonia in Dogs.—By the foregoing technic it is easy to produce pneumonia limited to one lung. It is even possible, by using a small amount of culture, concentrated by centrifugation to one-tenth the original volume, to produce pneumonia in one lobe, although this is much more difficult. From 10 to

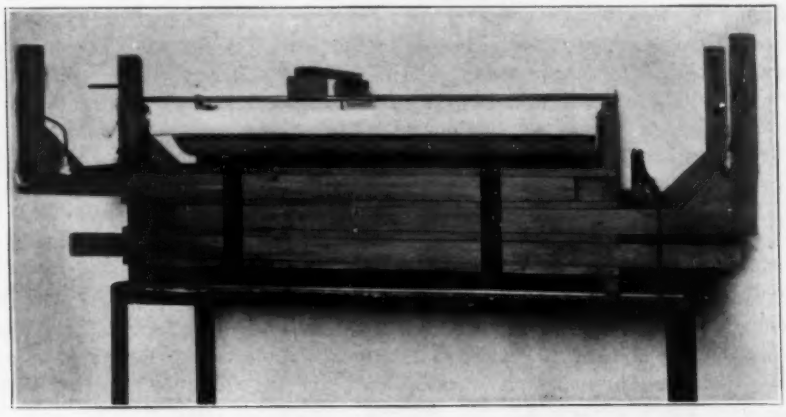

Fig. 1.—Special animal stand designed to secure symmetric roentgenograms; lateral view.

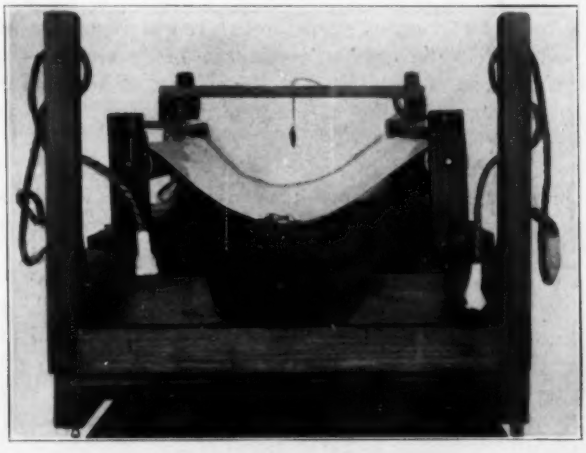

Fig. 2.—Same stand showing the lateral view of the shorter side.

15 cc. of the nineteen hour old pneumococcus culture previously described is usually sufficient to produce the disease.

The clinical symptoms in the dog and man are identical, but the diseases differ in their evolution and pathology. With the doses specified, bacteremia usually occurs in the dog on the second or even the first day; the animal may live only thirty hours, rarely longer than

three to six days after insufflation. When lesser amounts (5 cc. of nineteen hour old culture) were used or the culture was older than thirty hours, only evanescent infection set in, with slight clinical symptoms, the animal generally recovering in two or three days. The first observable signs of pneumonia in the dog were: diminished intensity of the respiratory sounds, a slight (rather tympanitic) dulness over the affected side, rise in temperature, dyspnea, cough and rapid pulse rate. The number of white blood cells generally increased at the beginning of the disease, varying from 8,000 to 20,000, with from 85 to 98 per cent of polymorphonuclears. In the toxic forms there was a rapid decrease to as low as 900 or 1,000 white blood cells, with practically 100 per cent polymorphonuclears.

When pneumonia was present, the roentgenograms invariably showed three definite features, namely, a haziness of the affected lobes, elevation of the diaphragm and displacement of the heart to the affected side. The consolidation was usually early, being manifest within from ten to fifteen hours. Dulness and bronchial breathing as a rule appeared later. Exceptional crepitation was noted. Moist or coarse râles were often heard in the nonaffected lung. Coughing, noticed in a majority of cases, was generally nonproductive and sometimes followed by vomiting. A purulent nasal discharge was noticed four times. The temperature was not always in accordance with the severity of the disease, and in toxic cases we generally observed a low fever or even hypothermia. Rapid pulse rate and respiration, a low white blood cell count, a low temperature (indicating great toxicity) and bacteremia were generally present.

Postmortem Observations.—One, two or more lobes were found involved. Usually only the insufflated lung was involved, but in a few instances there was an extension to the inferior lobe of the other lung, particularly when the nonconcentrated culture was used. In the latter cases the spread of infection was due, in all probability, to cough. The affected lobes appeared dark bluish, were much heavier than the healthy ones, had a fleshy consistency, were airless, and sank in water. Cut sections were more moist than those from human beings, and under compression gave a slightly frothy exudate, more or less depending on the age of the lesion; there was more of this type of exudate in the earlier stages, and it was diminished or absent twenty-four hours or more after the onset of the disease. At autopsy, immediately after death or after the animal was killed, the larger bronchi were filled with a thick viscid exudate and with a thinnish fluid when autopsy was postponed for even a few hours.

We wish to stress the fact that true "plugs" were not found in the large bronchi when an entire lung was involved. This would indicate

that complete occlusion of a bronchus may be accomplished by a sufficiently viscid fluid, possibly because of the existing lesions in the ciliated epithelium with consequent loss of function.

Histologic section shows that pneumococcic lobar pneumonia in the dog is not identical with that in man; it differs particularly in the

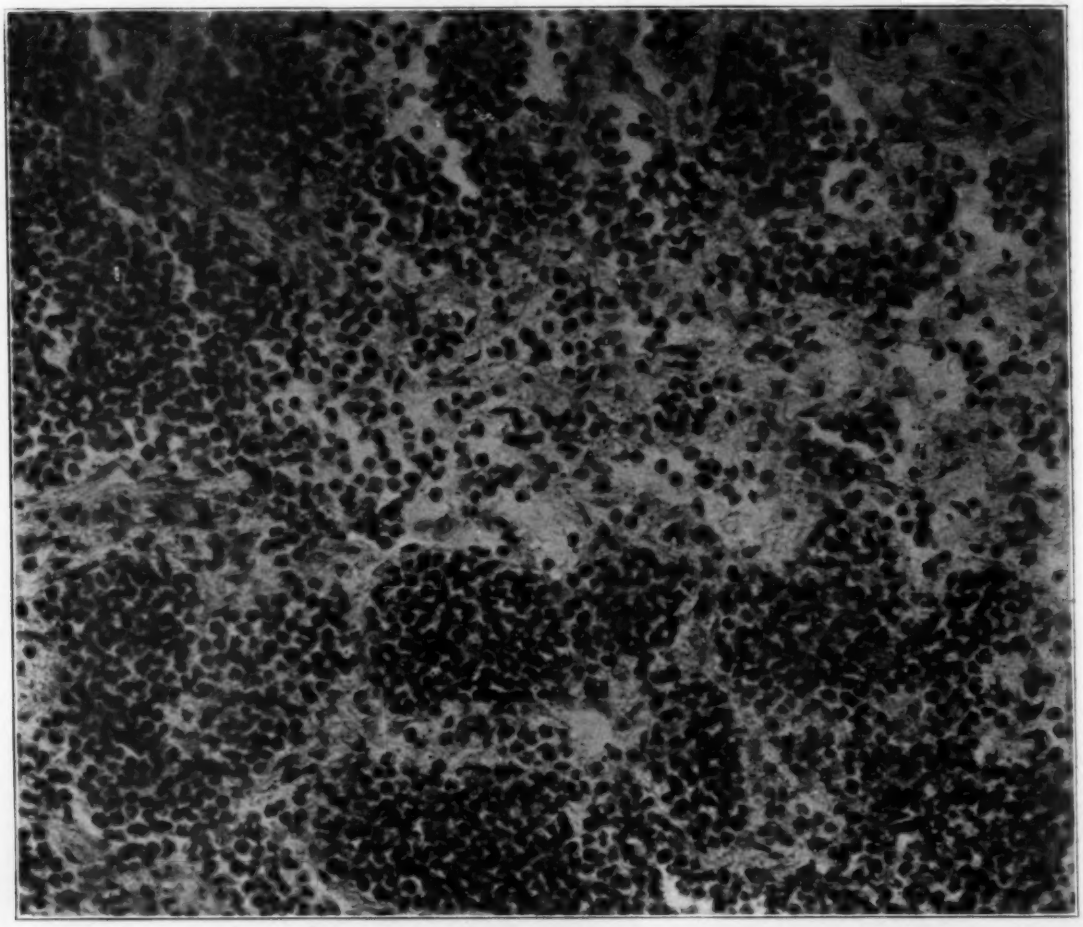

Fig. 3.—Photomicrograph of accessory lobe fifty-six hours after insufflation of the right main bronchus with pneumococcus culture; beginning gray hepatization; diplococci in exudate.

distinctly smaller amount of fibrin as compared to that in human beings (fig. 3).

Pleurisy, empyema and purulent pericarditis were observed, especially when the bronchus was obstructed with the balloon after previous insufflation of even a moderate dose of pneumococcus culture. Suppurative pneumonitis was produced once in a dog with an obstructed

bronchus and was followed by pyopneumothorax due to the opening of the abscess into the pleural cavity.

Forty-eight dogs were used: twenty-one were insufflated with pneumococcus culture; fifteen developed lobar pneumonia, and six showed slight or no infection. Of the fifteen, only two recovered and thirteen died. Twelve others were insufflated and then the bronchus blocked; all died. In six control dogs the bronchi were blocked without pneumococcus insufflation; the animals recovered completely, even when the balloon was removed after three days. One dog was given an intravenous injection of pneumococcic culture after a bronchus was blocked. Obstruction was established in another dog, and then a blood clot infected with pneumococcus was injected into its jugular vein. One was given an intravenous injection of anaerobes after bronchial obstruction, and one was given a straight intravenous injection of pneumococcus without previous blocking. Five dogs died so shortly after simple blocking of the bronchi that they are of no particular interest in this experiment.

Signs of Massive Atelectasis Accompanying Pneumonia.—In every one of our cases of experimental pneumonia in the dog, clinical symptoms and roentgen signs of atelectasis were present. We give as examples résumés of protocols of two cases—dogs B 27 and B 40. The first animal was insufflated with 10 cc. of pneumococcus type I culture, the second with the same amount of culture concentrated to 1 cc. by centrifugation.

Dog B 27.—Weight, 6 Kg. Blood count: white blood cells, 11,800 with 80 per cent polymorphonuclears.

Nov. 29, 1927: Intraperitoneal anesthesia. Narcosis complete in twelve minutes.

1 p. m.: Control roentgenogram (fig. 4 *A*) taken.

1 p. m.: Bronchoscope introduced and 10 cc. of a nineteen hour old culture of pneumococcus insufflated into left main bronchus.

5 p. m.: Dyspnea, nonproductive cough. Temperature, 98 F.; pulse rate, 104; respiration, 92. Base of left lung distinctly dull with absent breath sounds. Heart markedly displaced to the left. Roentgenogram shows lower lobe of left lung hazy; heart displaced to the left (fig. 4 *B*).

November 30: Very toxic, moribund; sphincters relaxed. Diarrhea present. Temperature, 94 F.; pulse rate, 96; respiration, 40. Dulness and flatness over the whole left side with bronchial breathing all over, except at the apex of left lung. Heart heard in left axilla. Right lung normal, with increased respiratory murmur.

10 a. m.: Roentgenogram shows left lung opaque; heart completely shifted to the left; left side of diaphragm elevated (fig. 5 *A*). This picture is practically identical with one of massive atelectasis of the left lung. Blood count: white blood cells, 2,400, 94 per cent polymorphonuclears.

12 a. m.: Bronchoscopy; 5 cc. of brownish, viscid fluid aspirated. Fifteen minutes later, another picture was taken showing only the base of the left lung possibly a trifle more aerated (fig. 5 *B*).

9 p. m.: Animal died.

Autopsy.—The trachea was clamped before the chest was opened. A small amount of thin, serosanguineous, pinkish fluid found in both pleural cavities. Right lung normal in color and consistency. Left lung, dark, bluish-black, has a fleshy feel, was friable, airless and sank in water. Cut section shows granular, dry surface, but expression of the tissue gave a sticky, thick fluid. The larger bronchi on the left side filled with viscid fluid. Affected lung is smaller than the healthy one. Typical case of a rapidly lethal pneumonia.

Dog B 40.—Weight, 9 Kg.

Dec. 15, 1927: Control picture taken.

2:30 p. m.: Intraperitoneal anesthesia. Narcosis complete in twenty minutes.

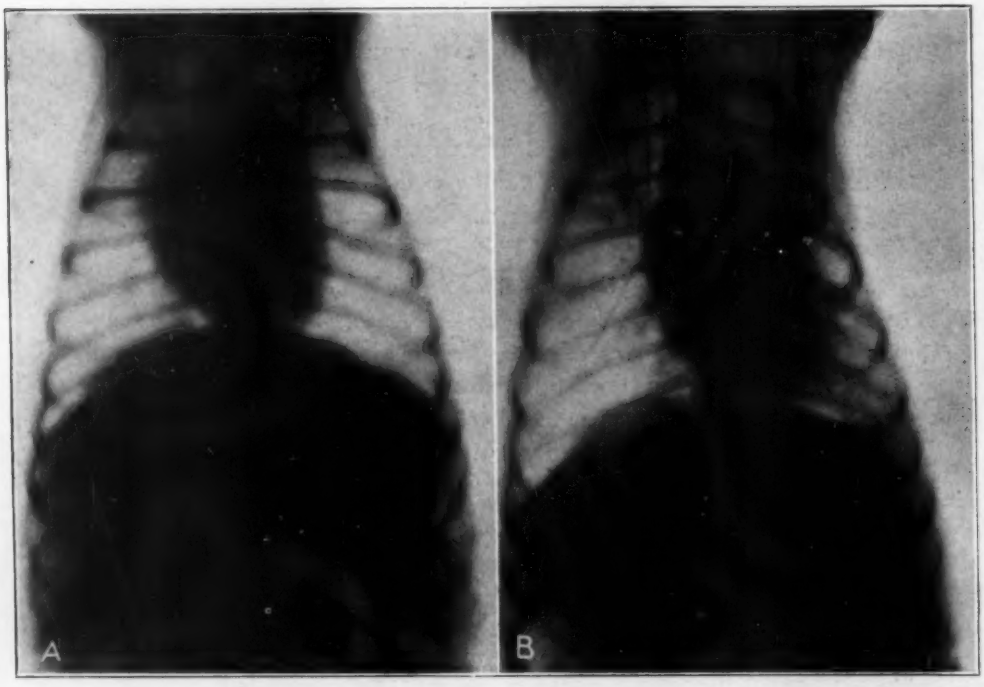

Fig. 4 (dog B 27).—*A* is a control roentgenogram; *B* was taken four hours after insufflation of 10 cc. of nineteen hour old broth culture of pneumococcus type I into the left main bronchus. Notice the displacement of the heart to the left and the haziness of the left lung.

3:10 p. m.: Bronchoscopy. Ten cubic centimeters of twenty-two hour old culture centrifugated the sediment taken up in 1 cc. of broth and atomized in the right main bronchus.

5 p. m.: Temperature, 96 F.; pulse rate, 120; respiration, 120.

December 16: Dog died at 9:30 a. m. Picture taken half an hour later showed complete consolidation of right lung; heart displaced to the right and right side of diaphragm elevated (fig. 6).

Autopsy.—The trachea was clamped. A very little pinkish fluid in the pleura. Left lung normal, somewhat overdistended. The affected lung smaller in size, dark bluish-black, airless, sank in water; appearance and consistency that of a lung in the stage of red hepatization.

Bacteriologic Examination.—Heart blood (taken aseptically), positive for pneumococcus. Pneumococci found in great numbers in alveolar exudate and pleural fluid.

These two cases represent very toxic forms of pneumonia. In the less toxic and more slowly developing forms, the degree of shifting of the heart and mediastinum depends on the number of lobes involved.

Influence of Mechanical Obstruction of a Bronchus Previously Insufflated with Pneumococcus Culture on the Development of Pneumonia.—It appears that mechanical occlusion of a bronchus previously

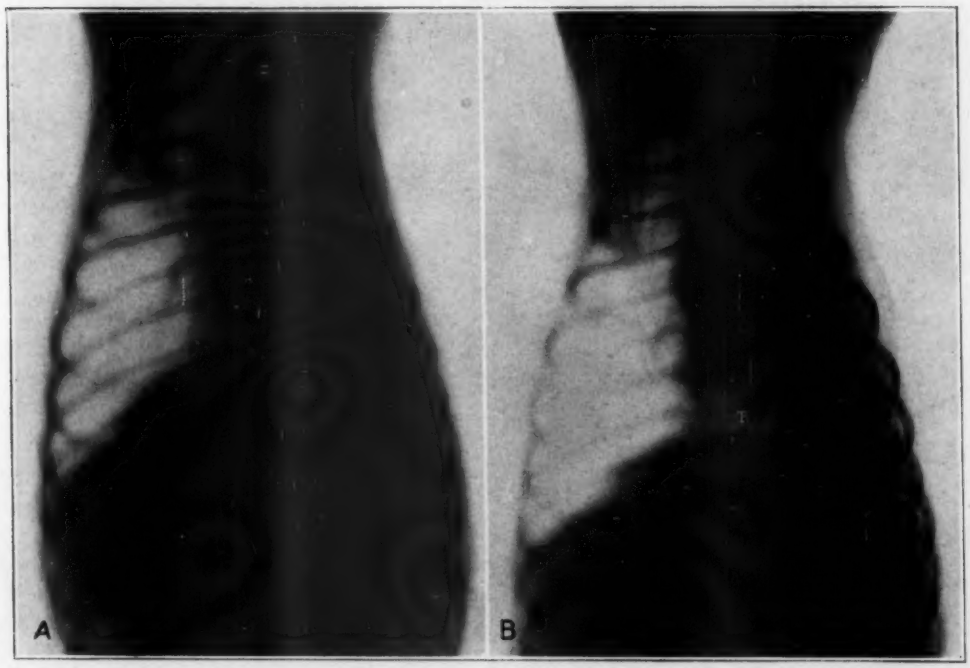

Fig. 5 (dog B 27).—*A* was taken twenty-two hours after insufflation of culture. The left lung is opaque; the heart is shifted to the left and the left side of the diaphragm is elevated. This picture is practically identical with one of simple obstructive massive atelectasis. *B* was taken twenty-four hours after insufflation of culture and fifteen minutes after aspiration of 5 cc. of viscid, brownish exudate from the left common bronchus. Note the clear triangular space *"T,"* common to pneumonia and atelectasis, described by F. T. Lord in the former, and due to encroachment of the healthy right lobe on the left thoracic cavity.

insufflated with pneumococcus culture increases the toxicity of the disease. Two dogs of the same size were insufflated the same day with equal amounts of the same culture, and in one of these the inferior bronchus was obstructed with a spongewood stick. We constantly noticed that when the culture was old and the amount was small (forty-

two to fifty-hour cultures—5 cc. amounts) the control dog developed only a mild, if any, pulmonary infection, whereas the "plugged" dog developed a severe and lethal pneumonia. When the culture was very virulent and the amount was larger (eighteen hour old culture—10 cc. or more), both dogs developed pneumonia, but the disease in the dog with the obstructed bronchus was much more severe and toxic.

We give here the protocols of four dogs: B 19, B 18, B 40 and B 41.

Dog B 19.—Weight, 6 Kg. Insufflation of culture without blocking. Recovery.

Nov. 17, 1927.—3 p. m.: Anesthesia by usual technic.

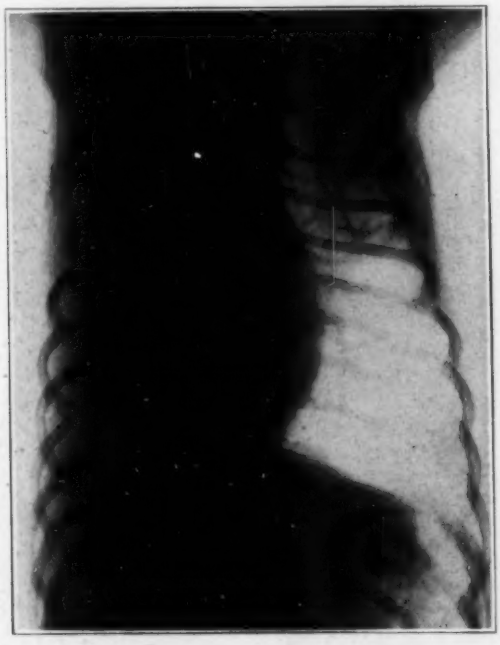

Fig. 6 (dog B 40).—Eighteen hours after insufflation of 1 cc. of culture (representing 10 cc. of twenty-two hour old centrifugated broth culture) into the right main bronchus. Notice the complete consolidation of the right side, the displacement of the heart to the right side, and the elevation of the diaphragm on the same side. This roentgenogram was taken one-half hour after death.

4 p. m.: Bronchoscope introduced. Five cubic centimeters of twenty-four-hour old broth culture pneumococcus type I insufflated into right common bronchus.

November 18.—11: 15 a. m.: Temperature, 103 F.; pulse rate, 180; respiration, 24. Coughs occasionally. Appears to be well. X-ray picture taken at 3 p. m. shows slight shifting of heart to the right, the right side of diaphragm higher than in control picture.

November 19: Same condition; temperature, 104 F.; pulse rate, 104; respiration, 20. Blood count: white blood cells, 12,500; 90 per cent polymorphonuclears. Roentgenogram shows no displacement of mediastinum. Blood culture negative.

November 21: Dog very lively. Roentgenogram shows heart in normal position. Temperature, 103.2 F.; pulse rate, 140; respiration, 24. Blood count: white blood cells, 11,700; 88 per cent polymorphonuclears.

November 22: Dog seems normal. Temperature, 102 F.; pulse rate, 120; respiration, 20. Chest normal. Blood count: white blood cells, 11,600.

November 23: Dog well. Sent back to its cage.

Dog B 18.—Weight, 6 Kg. Insufflation of culture with blocking. Death.

Nov. 17, 1927.—3:30 p. m.: Insufflation of 5 cc. of the same culture as was used for dog B 19, into the right lower bronchus; some contamination of the right common bronchus. Right inferior bronchus obstructed with a spongewood stick.

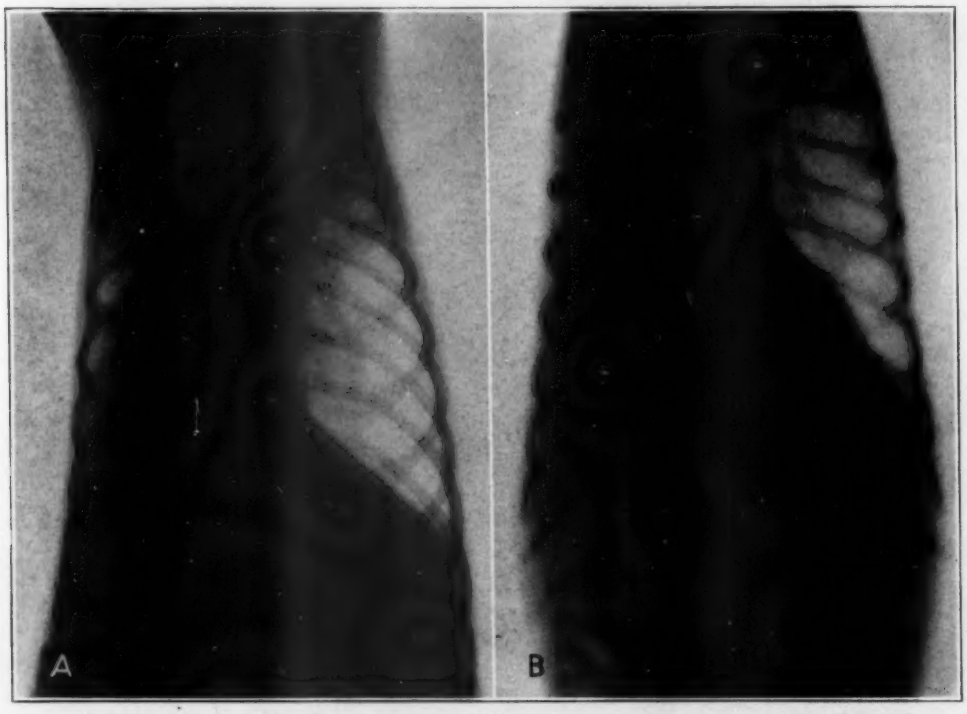

Fig. 7 (dog B 18).—*A* was taken twenty hours after insufflation of 5 cc. of twenty-four hour old culture of pneumococcus into the right lower bronchus which was immediately afterward obstructed with a spongewood stick. Notice the deviations of the heart to the right and the haziness of the lower and median lobes of the right lung. *B* was taken forty-four hours after insufflation of culture and blocking of the right lower bronchus. Empyema had developed and has displaced the heart to the left.

November 18: Temperature, 105 F.; pulse rate, 180; respiration, 64. Looks very sick. X-ray plate taken at 11 a. m. shows marked shifting of the heart to the right (fig. 7 *A*). Dulness and absence of breath in lower median lobes of right lung. Coughing slightly. Heart deviated to the right.

November 19.—11 a. m.: Dog very sick. Extremely dyspneic. Refuses food. Temperature, 104.4 F.; pulse rate, 180; respiration, 60. Toxic appearance in

marked contrast to that of dog B 19. Blood culture positive. White blood cells, 2,000; 95 per cent polymorphonuclears.

4:30 p. m.: Dog very sick and extremely dyspneic. Empyema (clearly visible in the roentgenogram, fig. 7 *B*) developed, and heart displaced to left side. Dog died same day.

Autopsy.—Empyema, consolidation of right median, inferior and subcardiac lobes.

Dog B 40 was insufflated with 10 cc. of culture concentrated to 1 cc. by centrifugation without blocking. Bronchus of dog B 41 was blocked with a balloon thoroughly dipped in the same concentrated solution so

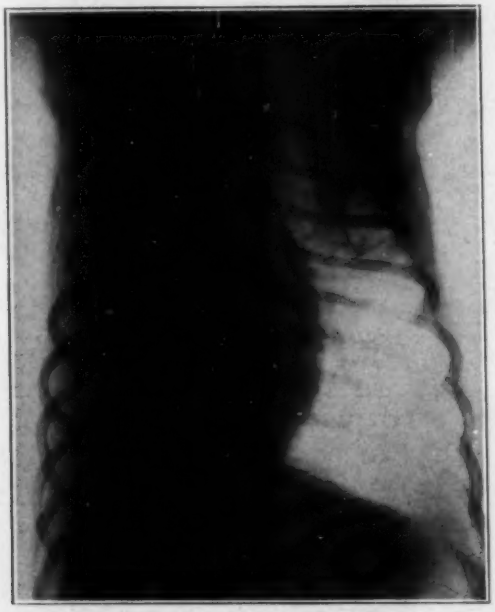

Fig. 8 (dog B 40).—Eighteen hours after insufflation of 1 cc. of concentrated culture one-half hour after death. The right lobe is opaque and the heart is markedly displaced to the right.

as to absorb an equal amount. The first animal died in eighteen hours; the second developed a fulminating form of the disease and died in twelve hours. The bacteriologic observations described hereafter are interesting.

Dog B 40.—Weight, 9 Kg.

Dec. 15, 1927.—3:10 p. m.: One cubic centimeter of ten times concentrated culture (nineteen hours' old) administered into the right common bronchus. Dog died eighteen hours later. A picture taken half an hour after death showed displacement of the heart to the right and an opaque right lobe (fig. 8).

Autopsy.—A very scanty mucoserous fluid in the pleura. Entire right lung consolidated and fleshy and sank in water.

Bacteriologic Observations.—Culture of heart blood negative. Cultures of pleural fluid and peripheral portion of right inferior lobe exudate positive for pneumococci. Smears of pleural exudate disclosed moderate numbers of polymorphonuclear cells but few diplococci. (One diplococci in several fields.) Smears of exudate from the peripheral part of the lungs showed very many diplococci in every field. It is interesting to compare these bacteriologic observations with those in the following experiment.

Dog B 41.—Weight, 7 Kg.

Dec. 15, 1927.—3:30 p. m.: Right common bronchus obstructed with balloon dipped in 1 cc. of concentrated culture twenty-one hours old.

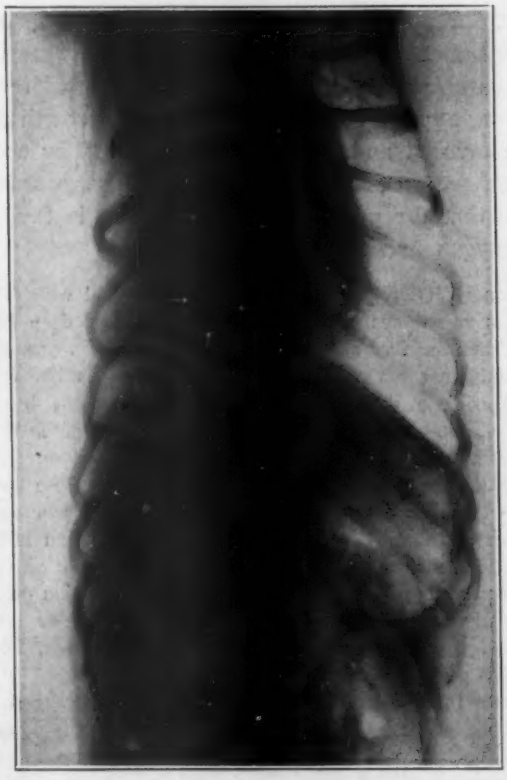

Fig. 9 (dog B 41).—Twelve hours after obstruction of the right main bronchus with an elastic balloon whose tip was dipped in a concentrated culture of pneumococcus before inflation. The roentgenogram was taken shortly after death.

5:30 p. m.: Roentgenogram showed a moderate shifting of the heart to the right.

December 16.—5:35 a. m.: Roentgenogram taken shortly after death. No marked displacement of heart to the right, probably because of rapid development of pneumonia and of pneumococcic cellulitis together with alveolar exudation before atelectasis was complete (fig. 9).

Autopsy.—Small amount of serosanguineous fluid in pleura. Balloon very well blown, in good position in right bronchus. The lower lobe of right lung uniformly consolidated; only patchy areas of consolidation in other lobes of same lung.

Bacteriologic Observations.—Heart's blood positive for pneumococci. Exudate in periphery of lower lobe of the right lung positive for pneumococci; pleural fluid positive for pneumococci; also present in great numbers on stained smear.

The differences between dog B 41 and dog B 40 are interesting. In the animal in which the bronchus was blocked (B 41), death occurred earlier, the heart blood was positive on culture and the pleural fluid and peripheral exudate of the involved lobe contained a much greater number of pneumococci. The deleterious effect of blocking a bronchus after insufflation of culture in the second instance was evidenced (1) by the more rapid spread of infection and the early appearance of great numbers of pneumococci in the periphery of the lung; (2) by the positive blood culture and (3) by the earlier death of the animal.

The foregoing experiments suggested that the growth of the pneumococcus may be favored and its virulence increased by obstruction of a bronchus. We have no definite evidence about increased virulence, but we have found that the growth of the organism is favored by partial reduction of oxygen tension.

In collaboration with Dr. J. C. Torrey, whose method we used, the following type of experiment was done: The bottom halves of two deep Petri dishes were selected so as to have the free edges fitting accurately when opposed. Into one of these was poured agar containing 10 per cent of rabbit's blood and 1 per cent dextrose. When cool, a definite amount of pneumococcus broth was surface stroked on this medium. In the other bottom half, plain agar seeded with *Bacillus subtilis* was poured. (*B. subtilis* may be surface stroked on the cooled agar.) For the control plate the plain agar was left untouched. The two halves or the two half dishes were then accurately opposed, the line of contact being covered with three thicknesses of adhesive tape over which two broad rubber bands were finally placed. The dishes were next incubated and periodic observations carried out. The plates containing *B. subtilis* gave sufficient reduction of oxygen tension, according to Dr. Torrey, to allow such organisms as *B. bifidus* (and other microaerophilic intestinal organisms as yet unidentified) to grow, by virtue of the oxygen consumption of *B. subtilis.*

Briefly, we have found that under such reduction of oxygen, the initial and subsequent growth of pneumococcus is favored; the growth is more luxuriant, and in many instances the individual colonies are larger and more succulent.

Influence of Removal of Bronchial Obstruction or of Aspiration of Bronchial Exudate on Development of Pneumonia.—Accurate concepts concerning this question are difficult to acquire, and our own experiments have not been very demonstrative as yet. There are two reasons for this: 1. When pneumonia develops in the dog, it is lethal. 2. The individual resistance of the animal to pneumococcic infection is so variable that it is difficult to interpret the effects of the relief of bronchial obstruction.

In dogs in which pneumonia was produced by an obstructing balloon dipped in culture, extraction did not produce the same striking changes

in the physical signs and clinical solution that occurred in experimental simple massive atelectasis. It seems as though the pneumonic consolidation had "fixed" the lung in its atelectatic condition, and removal of the obstructing balloon would therefore not give immediate or rapid aeration of the corresponding lobe.

Aspiration of the viscid exudate failed to give marked changes in the affected lobes except perhaps in one case.

In all our cases in which aspiration was performed, we obtained viscid brown or rusty fluid comparable to the human pneumonic exudate, but less rich in fibrin.

In the following case, pneumonia was produced by blocking the bronchus with a balloon dipped in twenty hour old culture, concentrated to one fifteenth of its original volume by centrifugation. The balloon was removed twenty-one hours later, and the common bronchus aspirated. Clearing of the lower lobe followed; the animal died eight hours later.

Dog B 38.—Weight, 6 Kg.

Dec. 13, 1927.—3:05 p. m.: Balloon dipped in 1 cc. of the concentrated culture introduced into the right common bronchus.

3:15 p. m.: Roentgenogram shows part of the median lobe atelectatic; heart shifted to the right, but right side of diaphragm not so high as might have been expected.

December 14.—10 a. m.: (Nineteen hours later.) Roentgenogram shows shadowing of the median and lower lobes of right lung; displacement of the trachea and elevation of the diaphragm; upper lobe, clear (fig. 10 *A*).

12:40 p. m.: Balloon removed and 15 cc. of thick brown exudate aspirated.

12:50 p. m.: Roentgenogram showed only a slight shadow persisting over median lobe (fig. 10 *B*). Animal died eight hours later.

We cannot interpret the clearing of the lower lobe as being entirely due to aspiration, because at autopsy a slight pneumothorax was found, possibly due to previous pulmonary injury while the heart was being punctured for a blood culture. Autopsy showed consolidation of the median and inferior lobes of the right lung. Pleurisy was not present. Blood culture was positive for the pneumococcus.

Production of Pneumonia by Transthoracic or Intravenous Injection of Pneumococcus Culture.—We have not been able to produce a pulmonary localization by transthoracic injection of pneumococcus culture into the lung.

Dog B 35.—Weight, 6.5 Kg.

Dec. 8, 1927.—1:45 p. m.: One cubic centimeter of twenty-two hour old pneumococcus type I culture was injected with a fine needle through the fifth interspace on the left side at the anterior axillary line. The needle was directed perpendicularly to the wall of the chest and penetrated 1.5 cm. The blood count showed: white blood cells, 6,300; 82 per cent polymorphonuclears. The following days the dog appeared to be fairly well with a temperature of about 103 F.

December 9: White blood cells, 17,250; 90 per cent polymorphonuclears.
December 10: White blood cells, 13,700; 92 per cent polymorphonuclears.
December 12: White blood cells, 5,000; 93 per cent polymorphonuclears.
All roentgenograms were negative for pneumonia.
December 15: Dog found dead.

Autopsy.—Tremendous amount of serosanguineous fluid filling both pleural cavities gushed out on opening of thorax. No sign of consolidation in collapsed lungs, which floated readily in water. Bronchial lymph nodes enlarged. No lesions in gross section of lungs. Pneumococci in pleural fluid.

We believe that the pleurisy may have been due to an indirect infection secondary to a bacteremia and only partly to direct infection of

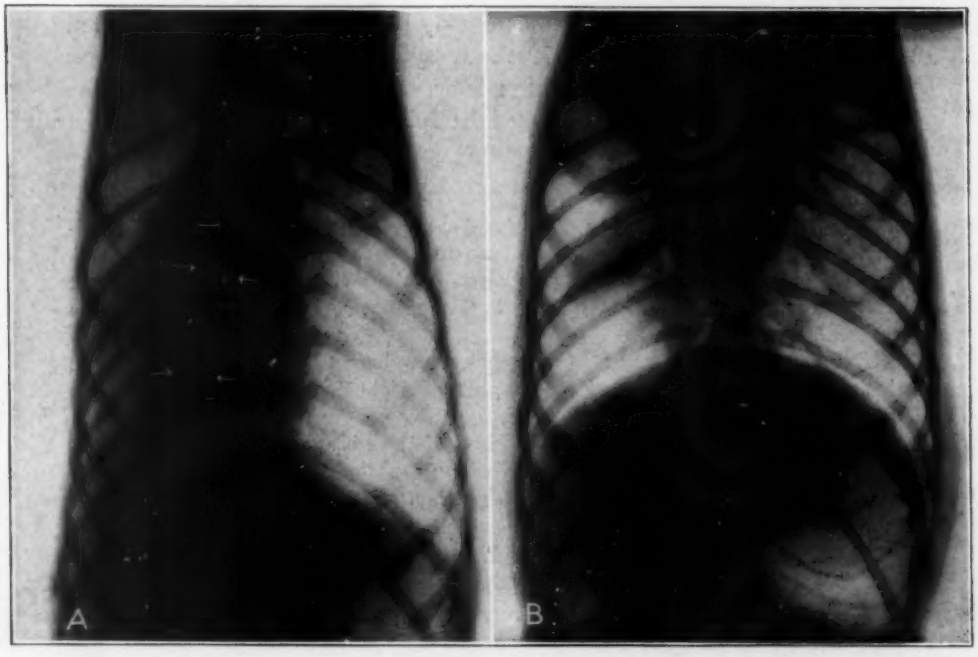

Fig. 10 (dog 38).—*A* shows an elastic obstructing balloon the tip of which was dipped in 1 cc. of concentrated culture and inflated in the right common bronchus. The roentgenogram was taken nineteen hours after this procedure. Notice the shadow over the lower and median lobes of the right lung and displacement of the heart to the right. The upper lobe of the right lung is clear. *B* was taken two hours and fifty minutes later and ten minutes after removal of the balloon and aspiration of 15 cc. of viscid, brownish exudate from the right common bronchus. There is a slight shadow over the median lobe of the right lung. The dog died eight hours later with partial pneumothorax.

the pleura. The delayed development of the pleurisy probably excludes direct infection of the pleura as a primary cause of the pleurisy. An illustration of a pneumococcic suppuration in the serous cavities with-

out localization in the blocked lung is given in dog B 43 in which a suppurative pericarditis developed after an intravenous injection of pneumococcus culture. Lesions did not develop in the control dog (not blocked) that received similar intravenous injection of a pneumococcus culture. Dog B 36 is the only animal in which a lobar pneumonia developed in an obstructed lobe following intravenous injection of pneumococci.

Dog B 43.—Right bronchus obstructed; twenty-two hours later, a complete right atelectasis was present (fig. 11). Intravenous injection of 1 cc. of pneumococcus culture, twenty-seven hours old. Balloon removed twenty-three hours later. The

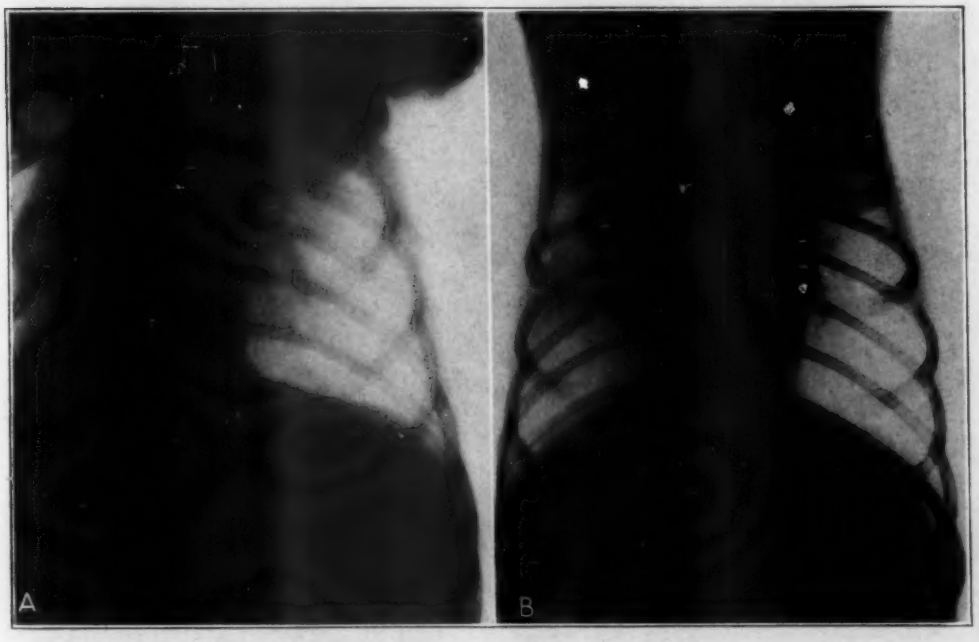

Fig. 11 (dog B 43).—*A* was taken twenty-two hours after simple obstruction of the right common bronchus. There is right massive atelectasis. One cubic centimeter of twenty-seven hour old culture was injected intravenously at this time. *B* was taken two hours after the removal of the balloon and twenty-five hours after intravenous injection of pneumococci. The heart has returned to the left and the right lung has cleared, but the cardiac shadow is somewhat enlarged.

heart returned rapidly to the left; the right lung cleared up, but cardiac shadow somewhat enlarged (fig. 11 *B*). General condition became gradually worse; severe toxic symptoms appeared. On the fifth day roentgenogram disclosed a markedly increased heart shadow (fig. 12). Animal died two hours later.

Autopsy.—Slight amount of fluid in the pleural cavity. Lungs normal. Pericardium tremendously dilated with approximately 250 cc. of light greenish pus. Heart coated with fibrin. Peritoneum normal.

Bacteriologic Observations.—No pneumococci in pleural fluid or on outer surface of pericardium. Pericardial pus loaded with white cells and innumerable diplococci. Culture of the heart blood ten hours before death was negative.

Dog B 47.—Right common bronchus blocked (fig. 13); nineteen hours later a blood clot infected with pneumococcus culture was introduced in the left external jugular vein. Twenty hours after this, the balloon was removed. The dog was very sick, with subnormal temperature. About forty-eight hours after the injection, no pulmonary localization was seen in the roentgenogram.

Autopsy.—Left lung normal. Right lung: right half of the subcardiac lobe partially consolidated (possibly an infarct). We were not able to locate the injected clot definitely.

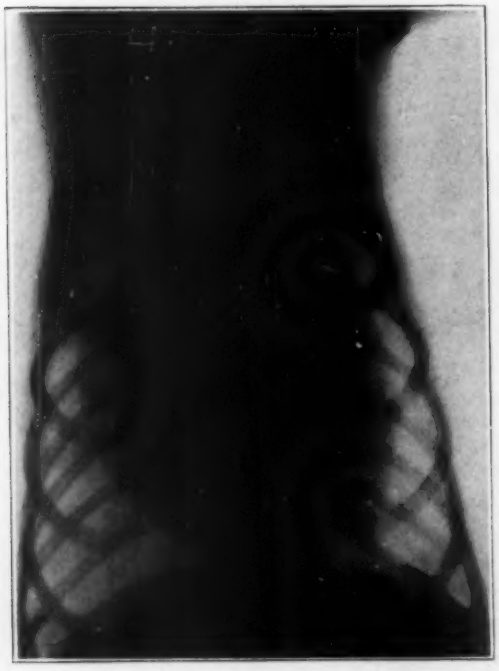

Figure 12

Figure 13

Fig. 12 (dog B 43).—Ninety-six hours after intravenous injection of pneumococci and seventy-three hours after removal of the balloon from the right common bronchus. Notice the markedly increased heart shadow which at autopsy proved to be a purulent pneumococcus pericarditis.

Fig. 13 (dog B 47).—Twenty-four hours after obstruction of the right common bronchus with a balloon.

The last three experiments have been cited because they suggest that massive atelectasis of the lung may have some bearing on the fixation in the lung or intrathoracic serous cavities of bacteria circulating in the blood. Lobar pneumonia was not produced by simple intravenous

injection of pneumococci or by transthoracic injection. Intratracheal insufflation of pneumococci, on the contrary, constantly produced lobar pneumonia.

ETIOLOGY

The bacterial incitants of acute primary infectious processes in the lung are well known. But, as A. Wadsworth [2] pointed out:

> Our knowledge of the essential conditions which determine the extent and nature of the disease processes in the lung lacks the precision which can only come from experimental data. This may be attained only when it is possible to reproduce at will the various disease processes in animals.

It is known, since the work of Prudden and Northrup, in 1889, of Silfast, in 1899, and of others, that the normal parenchyma of the lung, the mucosa of the bronchi and at least the lower part of the trachea are practically sterile (Hildebrandt, Rosenthal, Barthel, and others). The ciliated epithelium plays a predominant part in this sterilization, aided by the bactericidal qualities of mucus (Arloing) and by phagocytosis. In the mucous membranes of the nares and mouth, a great number of microbes are constantly harbored. Some of them disappear very rapidly; others may develop and exist as harmless parasites (Wright, Weininger, and others). Among them is found the microorganisms commonly associated with pneumonia, namely, pneumococcus (generally group IV), streptococcus, staphylococcus in 20 per cent of the bacillus and others. Netter found pneumococcus in 20 per cent of the sputums of persons who had never had pneumonia, and in 80 per cent of sputums of persons even several years after their attack of pneumonia.

Bezançon and Griffon found pneumococcus in 100 per cent of the forty tonsils they had examined.

Blake and Cecil proved that pneumococcus and *Streptococcus hemolyticus* introduced into the mouth and nares of monkeys cannot initiate an infection of the normal mucous membrane of the upper, much less of the lower, respiratory tract, even though the strains are very virulent. However, *Bacillus influenzae,* even when its virulence is not very great, can initiate in monkeys an acute infection of the normal mucous membranes of the upper respiratory tract, and this without the assistance of a preceding or concomitant contributing cause. Infection spreads readily to the lower respiratory tract, producing a tracheobronchitis and bronchiolitis, and permits the ready invasion of secondary bacteria, pneumococcus, streptococcus, etc. Thus may, to some extent, be explained the invasion of pathogenic microbes normally

2. A. Wadsworth's paper contains a full and accurate review of the literature on the etiology of pneumonia. We have borrowed liberally from it in the present study.

present as harmless saprophytes in the nares and mouth of healthy persons of the lower respiratory tract under the influence of a "common cold." The latter affliction prepares the ground by producing swelling and hyperemia of the mucous membrane, desquamation of the ciliated epithelium and infiltration of the mucosa and submucosa.

This mechanism explains the presence of the microbes of pneumonia in the lower portions of the respiratory tract, but cannot explain the production of a lobar or even a lobular pneumonia.

If the common bronchus of a dog or human being is opened, it will be noted that the orifices of the secondary bronchi going to the different lobes are close together; it is also known that they have a common blood and lymphatic supply. This being the case, why should the infection be localized in one lobe, to the exclusion of the remaining lobes of the lung? Further, when the disease subsides in one lobe and spreads to another territory of the lung, why does it again affect the same lobar character? This lobar distribution cannot be explained by the hematogenous theory of origin (because in that case the infection should spread all over the parenchyma of the lung) unless an infarct is assumed to be present—an assumption which is not supported by the pathology of lobar pneumonia. Even the bronchogenous theory of origin meets with the same difficulty of explaining the limitation of the process to lobar territories rather than lobular ones.

Few authors have faced this question squarely and still fewer have tried to find an answer to it.

Netter wrote in 1901:

> Pneumococcus does not suffice for determining a lobar pneumonia. Its arrival in the pulmonary parenchyma is not necessarily followed by lobar pneumonia. It may trespass the organ and localize in the pleura or pass into the circulating blood. It might even determine a broncho-pneumonia. *There must be some other factor which determines the localization of the disease right from the beginning and its limitation to one lobe.* In lobar pneumonia, before the beginning of the disease, the bronchi were healthy and the lung parenchyma normal. For the localization of the pneumococcus in one lobe, it is necessary that all parts of a lobe, to the exclusion of the other portions of the lung, be rapidly modified and a favorable soil formed for the development of the micro-organism.

Netter believed that cold exerts a reflex nervous action on a pulmonary lobe, but he added:

> There is no experimental proof of this fact, because irritation or section of the pulmonary nerves has never produced a true lobar pneumonia. But, unfortunately, there is no other way of explaining these two important facts, namely—limitation of the lesion to one lobe and onset of the disease very rapidly after exposure to cold.

In 1888, Lipari reported experimental results obtained in guinea-pigs and rabbits after introduction of pneumonic sputum into their

trachea. They were negative in the normal animals, whereas they were almost constantly positive when the animals had been kept for a while in ice water. He thought that the cold had an inhibitory action on the movement of the cilia, which, under normal conditions, are sufficient for protecting the lung against infection.

Bein gave the interesting history of a man, aged 30, who attempted suicide by throwing himself into the river; he was taken out immediately and transported to the hospital. He did not present any serious lesion or fever. One cubic centimeter of his saliva was injected into two rabbits, which died with pneumococcus septicemia. Six hours later the man's temperature rose to 102 F., he developed lobar pneumonia, and died on the sixth day.

Different theories of the action of cold in the development of pneumonia have been advanced. Cold may act on the tissues (1) by retention of deleterious products of body metabolism because of diminished excretions (Eisemann), (2) by reflex nervous action or (3) by direct action. The degrees of this action are largely dependent on the nature and extent of the exposure, the animal species and the nutritive condition of the person exposed. The marked fluctuations in blood pressure following exposure may give rise to hyperemia, congestion, transudation or even hemorrhage, and these changes predispose to many infections (Pasteur, Lode, Sanarelli, Kisskalt, etc.), but cold, by itself, cannot produce pneumonia (Heidenhain, Massalongo, Fischl, and others).

In the lung these circulatory disturbances are especially marked because of its anatomic structure. A great number of experimenters have tried to produce diffuse pneumonitis by introducing intratracheally various micro-organisms in animals previously exposed to cold. Only Lipari, who used virulent pneumococcus, reproduced lobar pneumonia. The others obtained only bronchopneumonia.

In the so-called "vagus pneumonia" (Muller), section of the vagi (unilateral or bilateral) is frequently followed by pneumonitis, usually of the patchy type, but sometimes of the lobar variety. Valsalva (1846), Schiff (1850) and others maintained that circulatory changes and especially paralysis of the larynx facilitate bronchogenous infection of the lung by saprophytic pneumococcus or other organisms. But as Traube (1846) and later, among others, Kline and Winternitz (1915) proved, pneumonia in these cases is due to paralysis of the larynx and aspiration of particles of food.

Traumatism of the lung was believed to be an exciting cause of pneumonia (Proust, Albert Koch, Reineboth, etc.). Undoubtedly in thoracic traumatisms, a series of changes can occur in the lung, even without opening of the thoracic cavity or direct contamination of the lung. For example, traumatism may predispose to massive atelectasis

and secondary pneumonia (Rose-Bradford, Coryllos and Birnbaum, etc.), but cannot be considered as a determining factor.

Internal traumatisms of the lung such as inhalation of various noxious substances in a gaseous, liquid or solid state produce a series of changes largely desquamative or exudative, as was shown by a great number of authors during the World War. These lesions, according to our theory, facilitate the accumulation of mucus in the bronchial tree and consequently the production of massive or patchy atelectasis, secondarily infected by the bacteria normally existing in the upper part of the respiratory tract.

Wadsworth injected pneumococci intratracheally into rabbits. In his exhaustive paper on "Etiology of Acute Pneumonitis" he admitted that the production of diffuse lesions in the normal rabbit is extremely uncertain and only possible "in the predisposed rabbit when both the general and local susceptibility are increased and when organisms of comparatively low virulence are used." He further added: "Organisms of low virulence induce evanescent bronchial reactions; more violent organisms give rise to more typical broncho-pneumonic lesions; while organisms of still greater virulence, if confined to the lung, incite diffuse processes of the lobar type." "But," he added, "the extremely fine balance of these conditions essential to the formation of lobar lesions in normal, as well as in predisposed animals, is yet beyond experimental control."

Wadsworth adopted a method of preliminary immunization of the rabbits so that very virulent cultures of pneumococci could be used without giving rise to bacteremic infection.

R. V. Lamar, S. J. Meltzer, M. Wollstein and their associates, experimenting on dogs and using intratracheal insufflation of pneumococcus, Friedländer's bacillus, streptococcus, staphylococcus, etc., concluded that the form of pneumonitis is chiefly influenced by the type of microbes insufflated, whereas the presence or absence of bacteremia depends on its virulence. Pneumococcus and often Friedländer's bacillus produced lobar pneumonia; the others, bronchopneumonia. Meltzer, further analyzing the action of pneumococcus, said: "The mere presence of pneumococci in the bronchi is not sufficient for producing a pneumococcus infection." But if the pneumococci are surrounded by tissue or inclosed in a canal or sac, they start to multipty and invade the surrounding tissues. "The same," he said, "occurs in diphtheria, where the infection and intoxication start when the bacilla are inclosed between necrotic membranes and the living tissues." He suggested that a similar situation may obtain where a "previous cold" furnishes a mucous secretion which "might occlude several small bronchi" and thus prepare a favorable ground for the pneumococci secondarily

infecting this mucous secretion to develop rapidly and invade the surrounding tissues.

Lamar and Meltzer thus came very close to our theory. They thought that "a few small occluded bronchi" could constitute a "start" for the intrapulmonary development of pneumococcus; but they did not go so far as to suspect the relation between the occlusion of a lobar bronchus by pneumococcic exudate and the development of "lobar pneumonia."

It is obvious that none of the foregoing causes—cold, circulatory changes, excitation or inhibition of nerve reflex, or even histologic changes—clearly and acceptably explains the anatomic localization and limitation of the lesion in lobar pneumonia. Any of these causes may, however, predispose to, and prepare the ground for, an inflammation of the bronchial tree, a tracheobronchitis and secondary infection by microbes normally or accidentally present in the upper respiratory tract.

Kline and Winternitz studied the effect of alcohol, ether and irritation of the respiratory tract with bromine fumes in animals, in which pneumococcus cultures were introduced into the larynx or the upper part of the trachea only. They came to the conclusion that "these factors seem to tend toward the development of a bronchitis or a bronchopneumonia both in animals receiving pneumococcus in the upper air passages and in animals not so treated."

From the foregoing it becomes clear that another factor is necessary for the production of lobar pneumonia. This factor is, in our opinion, the occlusion of a bronchus and the production of a "pneumococcic atelectasis."

PATHOGENESIS AND PATHOLOGY

The comparative study of the pathology of massive atelectasis and lobar pneumonia throws a new light on the latter disease.

Lobar Localization of Lobar Pneumonia.—The statistics show that in 100 autopsies in cases of pneumonia (Osler), the lesion occupied the right lung in 32, the left in 5 and both lungs in 17. The lower lobes were affected in 34 cases and together with other lobes in 27 cases, which gives them an incidence of about 61 per cent; the upper lobes were involved in only 13 cases. The statistics of Juergensen (in 16,614 cases), Biach, etc., give the same proportions.

Beddard stated that basal pneumonia occurs in about 75 per cent of cases; apical pneumonia in about 20 per cent, about 5 per cent being the so-called "central pneumonia." When two lobes are affected, it is most commonly both lower lobes. Double apical pneumonia is uncommon. Russell Cecil gives the same figures.

In healthy adults, the entire lung breathes with the same force. The accumulation of mucous exudate will then be more marked in the

more dependent bronchi and will simply be regulated by gravity. The right common bronchus and its division are more vertical than the left and their diameter relatively larger. Therefore, the expulsion of mucus is more difficult from the lower bronchi, especially those on the right. It is clear, then, we believe, that obstruction of these bronchi will be more easily realized, equally as well in lobar pneumonia as in postoperative atelectasis, or even postural atelectasis or pneumonia (in bedridden, exhausted, elderly patients); we think that this clearly explains the fact that the lower lobes are more frequently involved than the upper and the right more than the left.

In infants and elderly people, on the contrary, the upper lobes present less respiratory activity than the inferior, the respiration being more diaphragmatic than costal. This condition facilitates the accumulation of mucus and occlusion of the upper bronchi and consequently the production of apical pneumonia.

That the respiratory activity is of great importance in the determination of the bronchus to be occluded is shown in postoperative atelectasis, in which, as a rule, more than one lobe and often an entire lung is involved, generally on the side on which the patient lies. For in these cases the obstruction is due to the decrease of respiratory activity, absence of cough and splinting of the thorax because of pain, narcotics and posture.

Against this view is the fact, often mentioned in the literature, that no "plugs" are found at postmortem examination. This may be true, but it must be taken into consideration that autolysis of the fibrin in the exudate under the action of the proteolytic ferments continues after death, being a purely biochemical phenomenon, and that the "frothy exudate" which generally is found in the large bronchi may well be a modification of the one-time occluding exudate. Moreover, cutting open the bronchi disturbs the true intrabronchial conditions.

Osler stated that "the bronchi contain, as a rule, at the time of death"—or immediately thereafter—"a frothy serous fluid, rarely the tenacious mucus so characteristic of pneumonic sputum." One wonders what may have happened to this tenacious mucus which existed before death and about the origin of the "frothy serous fluid," for secretion certainly ceases after death. Furthermore, there have often been reported "fibrinous plugs in the larger tubes forming perfect casts," that a "thickish mucoid secretion may be found in the larger bronchi, while the bronchioles may be occluded by fibrinous plugs and cylinders of fibrin may be drawn out by forceps," (Osler) or that "the trachea and bronchi contain blood stained, viscid sputum," (Cecil). In the latter cases, we are led to believe that the proteolytic ferments could not have acted sufficiently on these fibrinous mucoid substances to digest them.

We are convinced that the bronchi corresponding to the consolidated area are obstructed by viscid exudate, basing our conviction not only on experiments on dogs, but also on bronchoscopic observations in cases of pneumonia in human beings. In every one of these cases, the bronchus corresponding to the affected area was found occluded by tenacious rusty exudate. Bronchoscopy has been performed in nine cases of pneumonia in human beings to date, and it is hoped that a greater number will soon enable us to report in detail the observations we have made in them.

Still another objection to our view of bronchial occlusion can be raised: If the bronchus is occluded, from where does the rusty sputum come? The objection is only apparent, because it would be difficult to maintain that this sputum comes from the alveoli or the terminal bronchioles of a lobe which has become rapidly consolidated. Besides, there can be no mucous secretion in the alveoli, in which no mucous glands exist, nor in the atria or terminal bronchioli; a few glands appear in the bronchioles having a diameter of 1 mm. or more. Therefore, the sputum must have its origin in the larger sized bronchi in which the mucous glands are numerous, their number and size increasing with the size of the bronchus. An overflow of mucus from the larger bronchi of the affected area may occur without necessarily disturbing the complete occlusion of these bronchi. In favor of this opinion is the fact that at the beginning the expectoration is usually scanty and increases as the disease advances, particularly at the period of resolution. If we compare the amount of sputum expectorated to the amount of exudate found in the involved lobes at postmortem examination, we will be convinced that an insignificant part of it is eliminated by cough during the period of red hepatization..

Inflammatory exudate in the alveoli, which is coagulated in situ, is the result of the rapid spread of the pneumococcic cellulitis from the hilum to the periphery, so that after the initial period of pneumococcic atelectasis there could rapidly follow a total obstruction of the broncho-alveolar spaces. Under these conditions, the mechanical difficulty of liberating the bronchi would render rather problematic the drainage of the affected lobe. We shall discuss this point further with the bronchoscopic treatment of lobar pneumonia.

Site of Origin and Mode of Spread.—Of great importance in the pathogenesis of lobar pneumonia is the determination of the point of origin of the disease. We shall not discuss the hematogenous theory of origin. A. Wadsworth, Lamar, Meltzer, Blake and Cecil, and others have experimentally proved the bronchogenous origin of the disease, whereas there is as yet no definite experimental proof of the hematogenous theory. "Experimental support of the hematogenous infection in

lobar pneumonia is certainly very meagre, uncorroborated and relatively insignificant," according to Wadsworth, in a review of the cases reported by Marchand, Thorner, Netter, etc.

Lamar and Meltzer and Blake and Cecil and the writers were not able to produce lobar pneumonia by intravenous injections of cultures, whereas they consistently did so in the dog or monkey by intratracheal insufflation.

The question arises whether the exudation and consolidation begin in the peripheral alveoli of the affected lobe and from there spread to the hilar portion or, inversely, start centrally at the hilum and spread to the periphery. This point has been thoroughly investigated by Blake and Cecil. They have proved that the lesion always starts at the hilar portion in the monkey. If for human beings Delafield and several of the older authors have upheld the other view, it is because they did not have the opportunity to follow up the evolution of the disease from its very inception, according to Blake and Cecil.

These authors stated:

> The most striking feature in the earliest stages, before hepatization, was a thickening of the walls of the bronchi near the root of the lobe, and of the vascular adventitia. This process spreads rapidly through the lobe, following the ramifications of the vascular and bronchial trees and appeared always to precede the exudation in the alveoli.

They further noticed:

> In monkeys that died early in the stage of red hepatization, the consolidation was found invariably to occupy the portions of the lobe proximal to the hilum. With the further progress of the disease, the distal portion of the lobe became progressively consolidated until complete lobar hepatization resulted. . . . The exudation appeared first in the parenchymal tissue, proximal to the hilum and subsequently involved the more distal portions.

The transition from red to gray hepatization characterized by infiltration of the exudate with white blood cells occurred first in the portion of the lobe in which red hepatization first developed, that is, in the parenchymal tissue proximal to the hilum. Another point of importance is the fact that very rapidly, often in the period of engorgement and before the lung is filled with firm exudate, the affected lobe becomes airless or atelectatic. We find further proof of this in the so-called "maladie de Woillez" of the French school, which is really nothing but a mild form of "lobar pneumonia." Woillez described this in 1848 as a clinical entity and Carriere has given a comprehensive clinical and pathologic description of it, although this disease, seldom fatal, offers only rare opportunities for postmortem study.

In a case of Carriere's in which autopsy was performed "the involved lobe was heavier than the unaffected one, had a beefy feel, the cut section showed a frothy, reddish fluid and the alveoli were filled

with exudate *containing no fibrin,* but a mucus staining pink with eosin, numerous red cells, desquamated epithelial cells, etc." This form of lobar pneumonia is almost identical with the "atypical form of post-operative pneumonia" described by A. O. Whipple, in which "the involved lobe or part of a lobe is heavier than normal, less air-containing, has a beefy feel and considerable pinkish fluid exudes from the cut surface. But hepatization as such is not seen, either grossly or microscopically."

Wollstein and Meltzer and the authors produced the same kind of pneumonia in dogs by intratracheal insufflation of nonvirulent pneumococci. The type of pneumonia described by Woillez, anatomically identical with the one due to insufflation of virulent pneumococcus, differs only from the latter in that the exudate is strikingly poor in fibrin and the consolidation tends to a more rapid resolution.

Thus, a number of points seem definitely proved. These are:

(1) The bronchogenous origin of pneumonia.
(2) The beginning of the lesion at the hilar portion of the lobe.
(3) The atelectatic appearance of the lobe from the start.
(4) The relation of the virulence of the pneumococcus to the amount of fibrin in the exudate.
(5) The relation of the amount of fibrin to the evolution of the disease, that is, the poorer in fibrin the exudate, the quicker the recovery is apt to be.

The Respective Size of the Affected and Unaffected Lobe.—In textbooks, the affected lobe is said to be of "a larger size than the unaffected one." This seems in contradiction to the elevation of the diaphragm on the affected side and the often found displacement of the mediastinum, heart and trachea to the same side, particularly noticed in children. What is more, displacement of the heart toward the healthy side, which should occur were the affected lobe really larger, has never been reported.

We firmly believe that this alleged increase in the size of the affected lobe is only apparent. In fact, after the chest and pleura are opened at autopsy and the intrathoracic negative pressure is eliminated (the trachea being patent), the healthy lung rapidly collapses, whereas the consolidated one is immobile and of the same volume as during life (fig. 14). But when we clamped the trachea before opening the thorax, we saw that the affected lung was smaller than the healthy one (fig. 15). We first suspected that the involved lobe is smaller from the roentgenograms taken of dogs and even of human beings (particularly children, as we shall elucidate later), which never showed displacement of the mediastinum to the opposite side. We confirmed these facts by observing the development of pneumonia in animals (with the chest open) placed in our box with "oscillating vacuum" (described in our paper on massive atelectasis). We thus clamped the trachea as a routine measure before

opening the chest. Further confirmation was had in the postmortem examination of a man, aged 60, who died on the seventh day of lobar pneumonia of the entire right lung, with the exception of two thirds of the median lobe. We first clamped the trachea before opening the peritoneal cavity and chest. The affected lung, although much heavier than the normal, was slightly but manifestly smaller than the left. We had occasion in this case to notice how rapidly the normal lung collapses, even with the trachea clamped, because of the rapid diffusion of the alveolar air through the walls of the peripheral alveoli.

Fig. 14 (dog B 15).—Anterior view of the heart and lungs. The left lung appears larger than the right with the trachea not clamped. The central light figure is the heart.

It may be well at this point to call attention to another factor which will tend to equalize the size of the healthy lung with reference to the pneumonic lung, as seen at postmortem examination, even with the trachea clamped, before the chest is opened. If a person dies with the lung in a phase of extreme expiration, there is a minimal amount of air in it. Add to this the previously mentioned diffusion of air through the alveoli of the sound lung, and the impression may be obtained that the healthy lung was not larger during life than the diseased one. Actually, during life the volume of the healthy organ must have been greater by at least the volume of the supplemental air.

Undoubtedly the relative difference in the sizes is not so striking as in simple massive atelectasis, because in "lobar pneumonia" the exudate partly takes the place of the absorbed air, whereas in uncomplicated atelectasis, there is very little if any exudate.

Resolution.—The pathologic process of this period is marked principally by the liquefaction of the exudate due to the digestion of the fibrin by the proteolytic ferment liberated by the white blood cells (Opie, Lord). Recovery from lobar pneumonia by crisis or lysis is

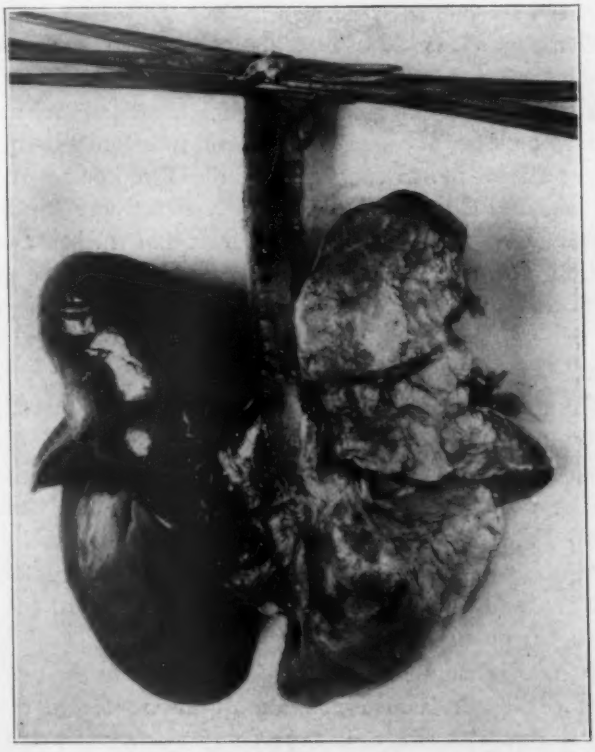

Fig. 15 (dog B 27).—Trachea clamped before opening the thorax. The affected lung is smaller than the healthy lung.

not quickly followed by disappearance of the lesion from the lung. The complete elimination by cough or absorption of the digested exudate takes from one to two weeks, as in such inflammatory diseases as ordinary abscess, appendicitis, etc. Time is necessary for the repair of the local lesion. Healing of the pulmonary lesion generally occurs within from ten to fifteen days after clinical subsidence of the disease, but if the drainage of the affected area is retarded, a delayed form of pneumonia may obtain.

Delayed or Unresolved Forms of Pneumonia.—O. C. Pickhardt, analyzing fifty-two cases diagnosed as "unresolved pneumonia," found only six which were really such. In two of these cases, in which the physical signs at the beginning are mentioned, we find "dulness diminished or absent fremitus, distant breathing." Curiously enough, in the roentgenograms of these six cases there is a characteristic sign of atelectasis, namely, displacement of the heart to the affected side, which did not seem to have attracted the author's attention. This displacement was not due to fibrotic contraction, because it disappeared gradually with the resolution of the pulmonary consolidation. In these cases, resolution was delayed apparently because bronchial obstruction was prolonged.

Even more striking are the pathologic similarities between atelectasis and so-called interstitial pneumonia first described by Charcot in 1878. In the massive or lobar form of this disease, most frequently following recurrent or delayed lobar pneumonia, the symptoms are those of massive atelectasis. Clinically, "there is a retracted and shrunken chest, the intercostal spaces are obliterated, the heart and trachea are displaced to the affected side, and dulness and absence of breathing are noticed over the affected area. The pathology consists of a frequent great shrinkage of the affected lobe with an airless and firm lung parenchyma."

There are all pathologic gradations, from a simple aseptic massive atelectasis to pneumococcic atelectasis, the sequence being (1) fetal lung or aseptic massive atelectasis, (2) mildly infected postoperative massive atelectasis, (3) postoperative massive atelectasis infected with pneumococcus group IV, (4) postoperative lobar pneumonia in its different forms, from the atypical group IV pneumococcus pneumonia to the more typical groups I, II or III and (5) at the other end of the sequence, the medical "lobar pneumonia."

It is easy to differentiate the forms of the disease at the two extremes of the sequence, but it is generally almost impossible to differentiate two adjacent forms when the only differences will depend on the nature and virulence of the infecting micro-organisms.

It would therefore seem that the pathologic changes in pneumonia strengthen our conception of "lobar pneumonia" as a pneumococcic lobar atelectasis.

CLINICAL EVOLUTION

Early Stages.—Lobar pneumonia and massive atelectasis have many clinical similarities. In fact, they differ only because of the special characteristics of the pneumococcus, that is, production of fibrinous exudate and toxicity. The onset in both diseases is sudden and generally without an apparent prodromic period, at least a very short one, usually occurring within twenty-four hours. It is accompanied by

dyspnea, cyanosis, rise of temperature and rapid pulse. Pleuritic pain is almost always present in both conditions. The initial chill and the toxic appearance of the patient, constant in pneumonia, are generally absent in atelectasis. The last two symptoms are due to the pneumococcus toxins.

The physical signs at the onset are similar as to (1) roentgen signs (the wedge-shaped shadow with its base on the periphery of the lung, the point directed toward the root of the affected lobe) and (2) stethoscopic signs (dulness, with rather a tympanitic quality, and the generally absent or distant breath sounds).

Besides the roentgen signs common to both diseases, there are other clinical similarities. The "dulness, with tympanic resonance and suppression of breath sounds, are of common occurrence in the earliest period of pneumonia. Quiet, suppressed breathing in the affected part is often a marked feature in the early stages" (Osler). "Suppression of breath sounds is an early symptom" (C. R. Austrian). "The earliest sign is weakness of the breath sounds over the affected area" (R. M. Leslie and J. B. Alexander), etc. Different explanations have been given: (1) start of the lesion at the peripheral portion of the bronchus, (2) deficient movement of the affected lobes or interposition of an aerated lung between the periphery and the bronchi. Only Pratt and Bushnell suggested as a possible cause "the plugging of a big bronchus with exudate."

These similarities in pneumonia and massive atelectasis at the onset can be explained, we believe, by but one mechanism, and that is the obstruction of a major bronchus by a relatively slightly infective exudate in atelectasis and by a highly virulent pneumococcic exudate in pneumonia. After the onset, the two diseases show more or less marked differences, depending on the toxicity and virulence of the micro-organisms present. In their typical forms, they look like altogether different clinical entities. Simple massive atelectasis usually resolves after from three to five days by lysis or crisis, with rapid and integral repair of the affected lung. The toxic element is practically absent. Clinically, we seldom find true bronchial breathing and crepitation (except in prolonged cases), absence of breath sounds (with or without râles) being the rule. The displacement of the mediastinum and its contents and the elevation of the diaphragm are much more marked in atelectasis than in pneumonia.

When, however, we come to the intermediate forms, these differences fade or even disappear. In the "mild" form of "post-operative pneumonia" it is impossible to draw a sharp distinction between the coexisting pictures of atelectasis and "mild" pneumonia.

The differences that exists between the typical and well developed forms of the two diseases are easily explained. The mucus in massive

atelectasis is slightly infected and almost exclusively localized in the larger bronchi; the alveoli are clear, so that after the absorption of the air in them there is a marked displacement of the mediastinum and diaphragm. The secretion in the bronchi is poor in fibrin and rather mobile, so that simply changing the position of the patient may induce a strong reflex cough which can disrupt or expel the "plug" from the bronchus. In pneumonia, on the contrary, the infecting microbe is highly virulent and a septic cellulitis rapidly spreads from the hilum to the periphery, accompanied by the production of a fibrinous inflammatory exudate. The consolidation of the affected lobe rapidly extends from the hilum to the periphery so that soon after the onset the whole lobe is affected. This explains the constancy of physical signs in pneumonia and the bronchial breathing due to interposition between the large bronchus and the periphery of a uniformly solid medium which transmits bronchial sounds to the ear.

Moreover, because of the rapid filling of the alveoli with inflammatory exudate, shrinkage of the affected lobe is less marked than in atelectasis; consequently, the displacement of the mediastinum and diaphragm is less marked or even absent.

During this period of evolution of pneumonia, an active growth and multiplication of pneumococci occur, probably favored by the decrease in oxygen tension, as we have already mentioned.

Crisis.—After a time generally varying between five and eleven days, the crisis occurs in pneumonia, the temperature drops, the patient rapidly recovers from the severe toxic symptoms and the lung gradually becomes normal. What is the cause of this rapid recovery? Is it the immunization of the body against the pneumococci—the destruction of the latter and the neutralization of its toxins by immunizing bodies developed in the blood? Or is it due to a local process such as liquefaction of the mucous exudate by the proteolytic ferments of the white blood cells and expectoration or absorption of the obstructing exudate and secretions?

Blake and Cecil remarked:

> At least a dual mechanism may be concerned in bringing about final recovery from lobar pneumonia. . . . There are cases in which septicemia persisted after crisis and disappeared after a while, and the animal recovered. On the other hand, in several instances fever came back after crisis and death occurred several days later due to a persisting septicemia. In another small group of cases, septicemia disappeared but crisis did not occur and the animal finally died, showing an unresolved pneumonia.

Another fact demonstrating this "dual mechanism" is the protection afforded against pneumococcic septicemia by previous injection of specific vaccine into the animal or human being. This immuniza-

tion does not protect against the local lesion. Wadsworth, Blake and Cecil, Kline and Winternitz, and others showed that intratracheal injection of pneumococcus cultures produce lobar pneumonia without bacteremia in previously immunized animals. Wadsworth immunized eleven rabbits by giving them injections, for other purposes, of pneumococci dissolved in rabbit's bile. These rabbits and five controls were given injections intratracheally of 1 cc. of virulent pneumococcus culture. The five controls died from bacteremia within five days and showed no localization of the condition in the lung. Of the eleven immunized, none died; postmortem examination disclosed more or less intensive massive consolidation. R. Cole has shown that septicemia in pneumonia may be averted in many cases by the administration of specific immune serum, but that the progress of the local lesion remains uninfluenced. Blake and Cecil immunized monkeys to pneumococcus by subcutaneous injections of pneumococcus vaccine. Although this induces humoral immunity and gives definite protection against septicemia from intravenous injection of virulent cultures of pneumococcus, it does not protect the monkey against lobar pneumonia produced by intratracheal injection of organisms.

These experiments are significant and demonstrate the dissociation between local and general infection in pneumonia.

It may be that previous progressive active immunization against the pneumococcus may limit the amount of fibrin in the exudate in the affected lobe; but it is certain, on the other hand, that liquefaction of the exudate and liberation of the bronchus and alveoli occur under the influence of other than immunizing factors alone. These are the proteolytic enzymes (leukoprotease, etc.) liberated by the white blood cells, and which have been thoroughly studied by Lockermann and especially by Ascoli and Benzola, Bittorf, Jobling and Strouse, Opie and his associations, F. T. Lord and others. In vitro, the pneumonic sputum left in the incubator at 37.5 C. for from twelve to eighteen hours loses viscosity and becomes very fluid because of the digestion of its fibrin. In vivo there is a biochemical interaction between ferments and antiferments; but because of the continuous influx of white blood cells, the proteolytic ferments are finally in the ascendancy. The phenomenon of the crisis occurring consistently on the fifth to the eleventh day may be due to this biochemical action rather than to biologic immunity.

F. T. Lord remarked:

The explanation of unresolved pneumonia is doubtless to be found in the chemistry of the exudate. The resolution is probably accomplished by local increase of cells (enzyme), diminution of serum (anti-enzyme) and shift to an acid reaction. Delayed resolution may be ascribed to a disturbance in the local ferment-

antiferment balance. If there is a shortage of cells or an excess of serum, resolution may be delayed or fail to take place.

It is difficult to explain by the theory of immunization alone the extension of consolidation to another lobe after more or less complete recovery of one lobe. Relapse after crisis, however seldom, is another strong argument against that theory of the mechanism of production of crisis.

It we admit that healing in massive atelectasis is due to the liberation of the obstructed bronchus, we are led to believe that the same mechanism must obtain in such a strikingly similar disease as "lobar pneumonia."

After an abscess, an infected gallbladder or the osteomyelitic focus are opened and drained, the temperature falls, the general condition improves and the toxicity diminishes. Why should pneumonia be an exception to this general rule? However extreme this surgical way of reasoning may seem, it must be admitted that it is more in accordance with the fundamental principles of general pathology than the local rôle attributed to immune bodies, which is as yet not definitely demonstrated. We are investigating this problem, and the results so far corroborate the foregoing opinions.

ROENTGEN OBSERVATIONS

Shape and Development of the Pneumonic Shadow.—Weill and Mouriquand, in a study of the roentgen signs in 350 cases of pneumonia, concluded that the early shadow is usually triangular, with its base always cortical. They have never seen a shadow which could be considered entirely central. They insist on the important fact that the shadow is often present long before the signs of consolidation appear; what is more, in some cases these signs may never appear.

Tripier also noticed the same phenomenon and stated that the consolidation always reaches the periphery of the lung and that the process is most intense in that region.

Mason, studying thirty-seven cases of uncomplicated lobar pneumonia in young children, found that the shadows were "always triangular in shape, with the base on some portion of the periphery of the lung; that from day to day they usually spread towards the root, gradually increasing until just before the crisis. In the early stages there is a relatively clear area between the apex of the shadow and the root of the lung. As the shadow spread, it obliterated the clear area until in most cases there was a uniform shadow extending across the lateral surface of the lung to the root."

According to Tripier, the process of consolidation seems to begin in that portion of the lung which is "anatomically farthest from the

hilum—that is to say—that part of the lung that lies close to the pleura, be it the parietal pleura, the mediastinal pleura, the diaphragmatic pleura or the pleura of the incisura." In all cases, bronchial breathing and bronchial voice did not appear until the shadow extended to the region of the root, because "only then a medium of comparatively uniform density is interposed between the site of production of the sounds (the trachea and large bronchi) and the point where the ear is applied."

Holt and Howland said that "consolidation occurs early, usually first at the periphery of the lung, gradually extending inward" and that often over the dull area there "may be so nearly an absence of all breath sounds as to suggest fluid." The same signs—wedge-shaped shadow and absence of sound over the affected area—have been noticed in the earliest stages of postoperative pneumonia (Allen O. Whipple, etc.).

But how can the appearance of the triangular shadow in the periphery and its gradual progression to the hilum be correlated with the well established fact that the infection in lobar pneumonia is bronchogenous, and that the pneumococcic cellulitis progresses from the hilum to the periphery?[3] If the shadow were due to the inflammation of the lung, it should appear to advance from the hilum to the periphery. Furthermore, its early appearance and the absence of respiratory sounds over the area corresponding to it seem inexplicable on the basis of our present knowledge about the pathogenesis of pneumonia. On the contrary, with the conception of lobar pneumonia as a pneumococcic atelectasis, the explanation of these signs becomes clear; every apparent difference between spontaneous and postoperative lobar pneumonia disappears. We believe, as previously stated, that the early shadow is due to the initial massive atelectasis, more accentuated in the periphery, because of the greater number of alveoli in this region, than at the hilum, where large-sized bronchi are present. The absence of sound is equally characteristic of atelectasis. The triangular shape of the shadow is due to the shape of the pulmonary parenchyma dependent on a bronchus. The simultaneous occurrence of pneumococcic inflammation which spreads from the hilum to the periphery explains the increase in size of the shadow and the appearance of bronchial sounds.

Displacement of the Mediastinum Toward and Elevation of the Diaphragm on the Affected Side.—The displacement of the organs of the mediastinum has generally been considered as a differential sign between atelectasis and pneumonia. L. Jaches stated that "massive post-operative collapse causes practically the same density of a lobe or the entire side of the chest as that seen in lobar pneumonia," and he

3. Even if we admit the hematogenous origin, we cannot explain the wedge-shaped shadow unless we admit a thrombosis of a big branch of the pulmonary artery. Such is not the case in lobar pneumonia.

added: "it may be distinguished from the latter by the displacement of the heart and the mediastinum to the affected side and by the comparatively larger size of the normal lung." However, in lobar pneumonia in children attention has often been drawn to the displacement of the trachea and heart to the affected side and elevation of the diaphragm on the same side. Thoenes, in 1922, gave the details in eleven cases with clinical symptoms of pneumonia (predominatingly in one lung) in which roentgen examination showed more or less marked displacement of the heart to the affected side. In four cases the diagnosis was confirmed at postmortem examination. He explained the displacement as due to overdistention of the healthy lung. Wallgren has observed many cases of pneumonia with displacement of the heart to the affected side, more particularly in early life (the first two and a half months to nine years). He concurred in Thoenes' explanation of the mechanism of its production. St. Engel pointed out that elevation of the diaphragm on the affected side in lobar pneumonia is of common occurrence. He is the only author who suggests that the displacement "might be dependent upon a diminution in size of the affected lung caused by a 'reflex interference' with respiration on that side."

J. P. Crozer Griffith, in his paper on massive atelectasis (1926), gave the history of a child less than a month old in whose case differentiation between atelectasis and pneumonia was extremely difficult. He considers that "it would seem that pneumonia may at times be capable of producing similar if not, perhaps, as marked, x-ray appearances as are seen in massive atelectasis . . . ." In a recent paper (1927), Griffith reported forty cases of lobar pneumonia in very young children. The disease predominated in one lung; sixteen exhibited displacement of the heart to the affected side. Griffith explained the displacement in the same way as do Thoenes and Wallgren—that is, hyperactivity and overdistention of the sound lung. He completely rejected the existence of atelectasis in these cases, without giving his reasons therefor, and considered the pneumonia responsible for this displacement of the mediastinum.

The explanation of the displacement of the heart and trachea by "compensatory hyperdistention of the nonaffected side" does not seem to us quite satisfactory. It is obviously impossible for the healthy and elastic lung to compress the resistant and consolidated affected lung to such an extent as to make displacement of the heart possible. Moreover, the pressure of the healthy lung should have a powerful lowering effect on the diaphragm, which, on the contrary, is found to be higher on the affected side. Displacement of the heart to one side of the thorax can be explained only by a decrease in the size of the diseased lung brought about by absorption of the air contained in the alveoli, with a resultant atelectasis. Atelectasis can occur only if the corresponding

bronchus is completely occluded. If these conditions are considered, the shifting of the heart and the simultaneous elevation of the diaphragm can easily be explained.

DIAGNOSIS

Diagnostic Difficulties in "Lobar Pneumonia" and Massive Atelectasis; Postoperative Pneumonia.[4]—For several reasons, these cases apparently resemble massive atelectasis more than do the medical lobar pneumonias. In the first place, the physician is at hand to observe the development of the disease in the postoperative case from its inception; this he can seldom do in cases of nonoperative pneumonia, as the patient generally enters the hospital from twenty-four to forty-eight hours or even more after the onset; in the second place, postoperative pneumonia has been closely studied during the past few years at the same time that massive atelectasis has been called to the attention of the surgeon. We will quote only a few authors who have written on postoperative complications of the lung, pneumonia or atelectasis in the past ten years.

Allen O. Whipple reported that in 1915 and 1916, in 97 of 3,719 patients operated on, pneumonia was diagnosed.

> The sputum was examined before and after examination, and in a great number of cases, serial roentgenograms were taken . . . daily notes were made of the chest, and if the patient showed signs or shadows of chest involvement, a blood culture was made, and if the preoperative and postoperative sputum specimen showed pneumococcus type IV, a specimen of the patient's blood was sent every third day for agglutination tests with the isolated pneumococcus strains, to determine the identity and grouping of the pneumococcus in its relation to the lesion found.

This paper contains data which are strong evidence in favor of our own contentions. After close scrutiny of the cases published as cases of postoperative pneumonia, we think we may state that every one was a case of postoperative massive atelectasis involving one or more lobes, secondarily infected with the pneumococcus and generally of the saprophytic group IV.

In fact, it is impossible from the histories of the mild or even of the more severe clinical types of cases to say whether they were cases of "postoperative atelectasis" or pneumonia. They present the characteristics of both these diseases and can therefore rightly be classified in both categories. In postoperative cases one finds that which is generally lacking in the "medical" cases of pneumonia, that is, observation at the very onset of the disease syndrome, so that the fusion process of atelectasis and pneumonia can be observed in a strikingly clear way. The etiology, onset, clinical symptoms, roentgen signs, evolution and

4. Postoperative atelectasis and pneumonia are more thoroughly studied in a forthcoming paper.

postmortem observations are typical of postoperative atelectasis, but bacteriologic examination reveals the group IV pneumococcus.

We give here a description of the "mild form" as given by Allen O. Whipple:

> The onset is usually sudden within the first forty-eight hours after operation, without an initial chill, but with a sharp rise in temperature. There is usually a moderate cough and at times a mild, pleuritic pain. The temperature seldom continues high, but within 24 to 48 hours begins to fall by lysis. *During the first few hours of initial high temperature, the radiogarms show a shadow in the lungs, usually in one of the lower lobes, frequently triangular or wedge-shaped.* At this time the physical signs are *dulness over the corresponding area with diminished breath sounds.* Bronchial voice and tubular or bronchial breathing necessary for diagnosis of consolidation, do not appear as a rule for twenty-four hours after the appearance of the initial shadow and after the drop of temperature. Rusty sputum is exceedingly rare in this type. The sputum, as a rule a yellow mucus, usually shows pneumococcus type IV in both the *preoperative and postoperative specimens.* Many of these cases show agglutination of either one or both of the pre-operative and post-operative pneumococcus type IV by the serum of the patient, taken from 7 to 14 days after the onset of the complication. This type occurs in otherwise healthy individuals as a rule, giving the history of a recent or concurrent "cold" at the time of the operation.

For the etiology of these cases, Whipple gave, among other causes: (1) local inflammation of the upper respiratory tract and factors favoring it; (2) factors inhibiting the normal thoracic and abdominal respiratory movements and favoring atelectasis and hypostasis in the lung.

The pathologic process found is, as we have already seen, equally characteristic of both diseases (atelectasis and pneumonia) in their earliest periods. Lately, in the service of the Second Surgical Division of Bellevue Hospital, one of us had an interesting case in which the patient had been operated on for a perforated gastric ulcer. Sixteen hours after operation he suddenly developed a typical massive atelectasis of the right lung. There was costal pain, dyspnea, shrunken chest, rise of temperature, dulness with absence of breath sounds and opacity of the right side of the chest in the roentgenograms, with displacement of the heart to and elevation of the diaphragm on the affected side. This patient was treated by rolling him on the sound side and thus causing violent coughing. He was cured by the fifth day. The sputum taken from him showed pneumococcus group IV in pure culture for several days after his recovery.

The question arises: What was this complication? Was it atelectasis complicated by the presence of the pneumococcus or a mild polylobar pneumonia in which the low virulence of the infecting pneumococcus made possible the early expulsion of the obstructing mucus? What would have happened had the pneumococcus been more virulent?

Would he not in the latter instance have developed a condition usually called "postoperative pneumonia?"

There is a striking resemblance between this "mild postoperative form" and the mild nonoperative form of pneumonia, and there is no doubt, as Whipple remarks, that the pneumococcus of "low toxicity" isolated by Carriere corresponds to our group IV.

In every one of the types of postoperative pneumonia the sequence of these phenomena is, in our opinion, as follows: (1) massive or patchy atelectasis; (2) secondary infection, generally with pneumococcus group IV, the saprophytic form which is present in over 50 per cent of healthy persons and in over 80 per cent of the cases which present this complication, and (3) development of a lobar or lobular pneumonia, unless the mucus obstructing the bronchus has been coughed out early, which generally happens because of the scarcity of fibrin in the bronchial exudate. In other words, the condition is massive or patchy atelectasis and infection with the pneumococcus.

According to the infecting agent, there may be a streptococcic, staphylococcic or even anaerobic or gangrenous atelectasis, the latter type generally following extensive operations on carcinomatous stomachs, the infection being due to aspiration of the foul content of the stomach ("deglutition pneumonia").

It is noteworthy that the embolic type is exceptionally rare. In 52 autopsies in a series of 143 cases of surgical pneumonia published by Henle, we find only 5 cases. In 7 of 25 fatal cases at the Presbyterian Hospital (A. O. Whipple), autopsy did not reveal embolism.

We shall mention one other common characteristic of both diseases, that is, predominance of the lesion in the lower lobes (almost 80 per cent) and particularly in the right lung (forty-seven as against thirty-five in the left). The x-ray picture in a case of postoperative pneumonia in the lower lobe of the right lung, given in Whipple's paper (bronchi injected with iodized oil), shows clearly complete obstruction of the main inferior right bronchus, the iodized oil stopping sharply at its origin. If we bear in mind that, as Whipple reports, generally the x-ray shadow appears before any physicial signs of consolidation are elicited, we may conclude, we believe, that the obstruction of the bronchus supplying the affected lobe is not secondary to the consolidation, but precedes it and is the immediate cause of it.

To summarize, this study of the postoperative cases demonstrates the relation of massive atelectasis to lobar pneumonia and patchy atelectasis to lobular or bronchopneumonia, the anatomic extent of the disease depending on the size of the bronchus occluded.

Besides these cases, there are a great number of cases of postoperative pneumonia reported in which the sequence, postoperative atelectasis—lobar pneumonia, is absolutely clear. L. G. Rigler reported a number of postoperative complications of the lung. A résumé of

two of these cases will show better than any other argument that it is impossible to draw a sharp dividing line between the two diseases.

CASE 4.—A bilateral salpingectomy was performed on a middle-aged woman. Twenty-four hours after operation, the temperature rose to 100 F., and cough appeared. The next day the temperature was 101 F.; there were cyanosis, cough and signs of consolidation of the right lung. Roentgen examination showed the right lung opaque and the mediastinum markedly displaced to the right. The temperature rose to 104.2 F., with a blood count of 15,000 white cells. Symptoms continued for ten days after the onset, when the crisis took place, following which roentgen examination showed resolution but retraction of the heart still present. After two days, the roentgen examination showed the lung to be clear and aerated and the heart in normal position. Symptoms of empyema then developed and were confirmed by the roentgenogram which showed the heart displaced to the left.

CASE 5.—The patient had undergone a salpingectomy. The onset and clinical course of her condition were typical of massive atelectasis until the third day after the onset, when rusty sputum appeared, and there were distinct signs of consolidations of the upper lobe of the right lung. Roentgen examination the next day showed upper lobar pneumonia. In addition, there were haziness of the median and lower lobes of the right lung, marked displacement of the mediastinum to the right and very high diaphragm on the right side.

The author concluded that in these cases lobar pneumonia was superimposed on a postoperative, massive atelectasis. On the other hand, we found in the literature a great number of cases of atelectasis described as complicating lobar pneumonia.

We give here the résumés of three cases published by N. B. Gwyn in 1926.

CASE 1.—A woman, aged 60, had complete consolidation of the lower lobe of the right lung. About the fifth day of the disease, she was suddenly seized with dyspnea, prostration and cyanosis. The heart beat was rapid and weak. The upper lobe, which formerly overexpanded, apparently was not solid. Physical signs were those of a newly consolidated area. The heart was displaced, the apex beating to the right border of the sternum. There was no cardiac dulness to the left side of the sternum. Clearing up began in six hours, and the heart returned to its normal position in four days.

CASE 2.—The patient entered the hospital with signs of a small area of consolidation at the base of the left lung. During the night there occurred an attack of dyspnea, and in the morning the involvement seemed more extensive. There was marked displacement of the heart, the apex being felt in the left axillary line, and the left side of the chest was flatter than the right. Roentgen examination showed a typical picture of atelectasis. Healing occurred after a week, with return of the mediastinum to its normal position.

CASE 3.—A boy, aged 6, had pneumonia of the lower lobe of the left lung. Involvement of the apex occurred at the end of two weeks without any added distress. The chest was completely solid; the sounds were tubular, but varying; from day to day the presence or absence of râles was noted. The left side of the chest was sunken and motionless. The right side was overextended and clear. The heart was displaced to the left and upward. The left side of the diaphragm was elevated to the level of the third rib in front. The left lung was very solid behind.

Fluid was not obtained on puncture. Three days later, examination showed that resonance was appearing in the midback and that the heart was lower in the chest. Roentgen examination revealed the heart in place, the diaphragm lower.

The foregoing three instances are described by Gwyn as cases of atelectasis complicating pneumonia.

These facts show clearly that the diagnosis between massive atelectasis and lobar pneumonia is often impossible. Atelectasis complicating pneumonia and pneumonia complicating atelectasis have both been reported. Furthermore, atelectasis and pneumonia are often found

Figure 16

Figure 17

Fig. 16 (dog 251).—Left massive atelectasis produced by obstruction of the left main bronchus with an "occluding balloon" twenty-four hours previously. Note the clear triangular area "T."

Fig. 17 (dog B 27).—Left "lobar pneumonia" (pneumococcic massive atelectasis) twenty-four hours after insufflation of 10 cc. of nineteen hour old broth culture of pneumococcus type I into the left main bronchus. Compare with figure 16.

together, and the same case is diagnosed "pneumonia with signs of atelectasis" or "atelectasis with symptoms of pneumonia."

We believe that these difficulties are due only to the fact that the close relationship between the two diseases has not been seen. They

disappear if we concede that lobar pneumonia is a "pneumococcic atelectasis."

TREATMENT

We believe that the general infection (bacteremia) in lobar pneumonia is a distinct and separate entity from the local lesion to which it is secondary. In their experimental study of prophylactic vaccination against pneumococcus pneumonia in monkeys, R. S. Cecil and F. Blake came to the conclusion that "subcutaneous vaccination with pneumococcus vaccine gives definite protection against experimental pneumococcus septicemia, but it does not protect against intratracheal infection. In vaccinated animals, the course of the disease is modified, the proportion of recoveries being considerably higher than for nonvaccinated monkeys."

Fig. 18.—Left massive atelectasis in man, postoperative. Notice the deviation of the trachea to the left and the absence of the heart shadow to the right of the vertebral column. Compare to figures 16 and 17. (Courtesy of Dr. Belden, New York Hospital.)

Because of the dual entities seemingly present in pneumonia, and of the excellent results obtained so far in the treatment of persons with postoperative massive atelectasis or atelectasis due to foreign-body

obstruction, it occurred to us that the relief of the mechanical bronchial obstruction possibly present might be of value in the treatment of patients with pneumonia.

This problem is far more complicated in pneumonia than in post-operative atelectasis because of the fibrin present in the pneumococcic exudate and the toxic element. The exudate or secretion in post-operative atelectasis is more fluid, has no marked tendency to reform, and the alveoli are not filled with it. It is rather confined to the large bronchi. In pneumonia, on the contrary, the exudate is

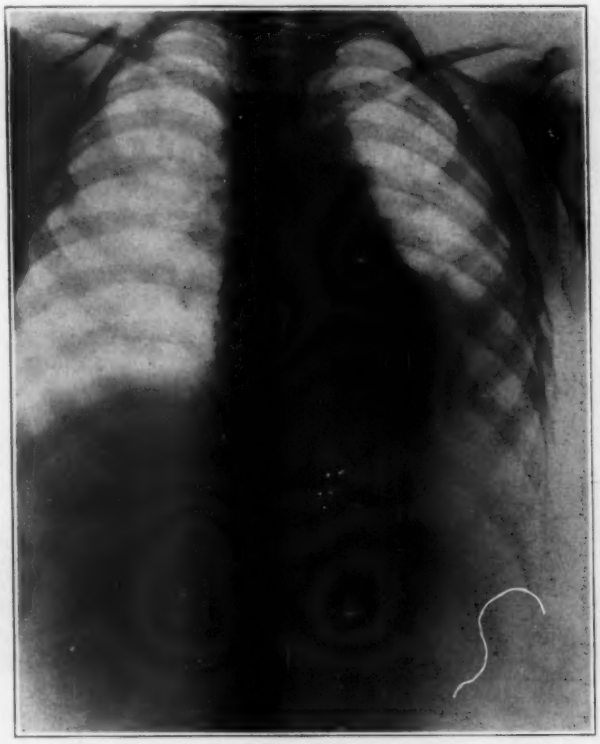

Fig. 19.—"Lobar pneumonia" in man in lower lobe of left lung. Notice the deviation of the trachea to the left, the haziness of the lower lobe and the heart drawn to the left. It should be borne in mind that but a single lobe is involved when comparing this figure to figure 18. (Courtesy of Dr. Belden, New York Hospital.)

extremely tenacious; it is continuously reformed and coagulates in the alveoli. It is possible to aspirate the large bronchi and the second and third order bronchi, but not the small bronchioli and alveoli.

It seems reasonable to suppose that the solution of this problem may lie in a combination of local and general treatment: (1) bronchoscopic aspiration for the possible liberation of the larger bronchi,

even if only temporary (and creation of an airway in them, and (2) antibody treatment against the general infections (bacteremia).

Theoretically, bronchoscopic aspiration in pneumonia could be effective only at the earliest period of the disease, the atelectatic stage (if we may so call it), when there is not yet a generalized consolidation in the peripheral bronchioli.

Practically, it is almost impossible to see early cases in the hospital wards unless pneumonia develops during the hospitalization of the patient, as in "postoperative pneumonia."

Figure 20

Figure 21

Fig. 20 (dog B 47).—Right massive atelectasis twenty-four hours after obstruction of the right main bronchus. Notice the shifting of the mediastinum to the right, haziness of the right lung and elevation of the diaphragm to the same side.

Fig. 21 (dog B 40).—Right "lobar pneumonia" (pneumococcic massive atelectasis) eighteen hours after insufflation of 1 cc. of centrifugated (concentrated) nineteen hour old pneumococcus culture into the right main bronchus. Compare this with figure 20.

Notwithstanding these many difficulties, we submitted the question to Dr. Lewis A. Conner at the New York Hospital and Dr. Alexander Lambert at Bellevue Hospital. With the collaboration of Dr. J. D. Kernan as bronchoscopist, we decided to submit our idea to a clinical

test. Bronchoscopy has been performed in nine cases so far—three in the New York Hospital and six in Bellevue Hospital, without any further modification of the routine treatment received by the patients in the respective services.

The number of tests is too small to enable us to draw any definite conclusions at the present time. We can, however, state that bronchoscopy did not produce the slightest shock or untoward effect, although some of the patients were old and very toxic. On the contrary, as a rule the patients claimed to have been more or less relieved of the local symptoms by the procedure; and the expectoration was considerably increased in some immediately afterward. The amount aspirated varied

Fig. 22.—Right massive atelectasis in man, postoperative. Compare this to figures 20 and 21. (Courtesy of Dr. Belden, New York Hospital.)

from 1 to 20 cc. The exudate was always extremely tenacious and aspirated with difficulty.

The course of the disease varied so in the different cases as to make it impossible to formulate conclusions. Although very encouraging in a majority of the cases, the results of aspiration have been disappointing in others. So far no deductions can be drawn regarding the influence of bronchoscopic aspiration on the evolution of the disease. We are now studying this treatment and the results obtained will be given in a subsequent paper.

We are purposely placing together, to facilitate comparison, eight roentgen reproductions of atelectasis and pneumonia, both experimental and clinical. The legends make these self-explanatory.

CONCLUSIONS

1. A new conception of pneumonia has been presented, based on experimental and clinical data.

2. Lobar pneumonia is considered as an "infectious (generally pneumococcic) lobar atelectasis of the lung."

3. Postoperative massive atelectasis, postoperative pneumonia and "lobar pneumonia" are shown to have a similar pathogenesis and evolution, and clinical and roentgen signs.

Fig. 23.—"Lobar pneumonia" in man in the lower lobe of the right lung. Notice the moderate shifting of the heart to the right, the haziness of the lower lobe and the elevation of the right side of the diaphragm. A marked shifting of the mediastinum is not to be expected when but a single lobe is involved.

4. Bronchopneumonia is considered as an infectious patchy atelectasis.

5. Bronchoscopic treatment, which has given such encouraging results in massive atelectasis, has been suggested and applied to cases of lobar pneumonia in man. The number of these cases is as yet too small to allow definite conclusions to be drawn from them.

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EXPERIMENTAL ATELECTASIS

WALTER ESTELL LEE, M.D.

GABRIEL TUCKER, M.D.

I. S. RAVDIN, M.D.

AND

EUGENE PENDERGRASS, M.D.

PHILADELPHIA

At the meeting of the Philadelphia and New York Academies of Surgery [1] in February, 1928, we reported the reproduction of massive atelectasis in a dog after injecting into the main bronchus of the right lung the obstructing bronchial secretion obtained by bronchoscopic drainage from the main bronchus of the left lung of a patient suffering from this condition (supplied by Dr. Clerf).

That bronchial obstruction is a cause of atelectasis was shown experimentally by Mendelsohn, in 1841, and again by Lictheim, in 1878. The work of Chevalier Jackson with foreign body obstruction of the bronchi has established the clinical phases of this phenomenon beyond all possibility of controversy. The similarity of the atelectasis produced by foreign body obstruction, described by Jackson, to that of postoperative massive atelectasis suggests similar causative factors. Elliott and Dingley, in 1914, accepted this explanation of obstructive atelectasis, and Leopold and Lee, in 1924, Churchill, in 1925, Mastics, in 1927, and Coryllos and Birnbaum, in 1928, agreed "that complete bronchial obstruction offers the best explanation for the cause of this condition." But most of the contributors to the subject are unwilling to accept this.

We have been encouraged by Dr. Willy Meyer in the hope that this Association will be interested in the experimental atelectasis we are producing in the Laboratory for Surgical Research at the University of Pennsylvania. Though previous attempts to produce experimental atelectasis by foreign body obstruction have been successful (Mendelsohn, Lictheim, and the recent exhaustive and convincing work of Coryllos and Birnbaum), we can find no record in literature of the use of the obstructing bronchial secretion removed from a clinical case of postoperative pulmonary atelectasis to reproduce the lesion in animals.

That we have succeeded in thus reproducing the lesion, by employing a synthetic material the physical properties of which are simi-

1. A report of this meeting appears in Ann. Surg. **88**:6 (June) 1928.

Fig. 1 (C. M.).—Massive atelectases of the left lung twenty-four hours after radical right herniorrhaphy. (From the Pennsylvania Hospital; Mitchell-Lee-Clerf-Bishop.)

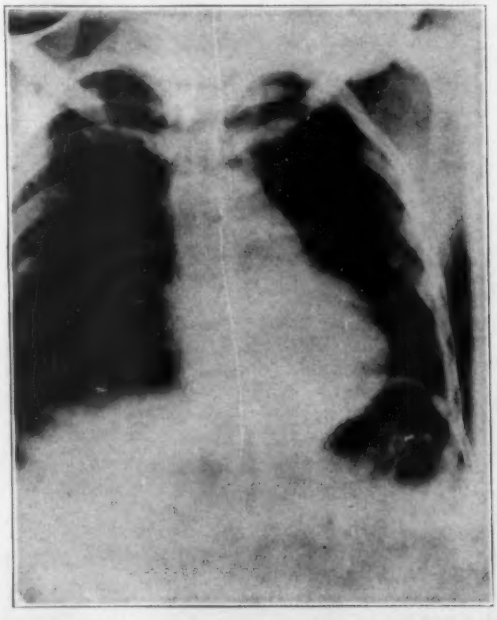

Fig. 2 (C. M.).—Twelve hours after bronchoscopic drainage of obstructing secretion from main bronchus leading to atelectatic left lung; complete recovery eighteen hours after onset of clinical symptoms. (From the Pennsylvania Hospital; Mitchell-Lee-Clerf-Bowen.)

lar to the bronchial secretion removed from this patient, is due, we believe, to our ability to abolish the cough reflex in our dogs. We are convinced that previous failures to reproduce this condition has been due to the inability to control the cough reflex. The cough reflex is notoriously efficient in dogs, so efficient that Jackson and his staff have found

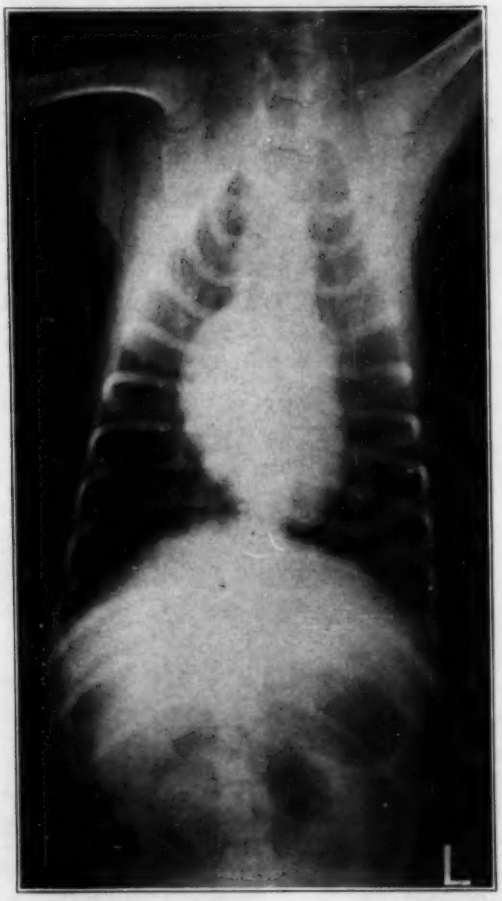

Fig. 3 (dog 456).—Twenty-four hours before experimental production of massive atelectasis. (From the Department of Surgical Research, University of Pennsylvania; Lee-Tucker-Ravdin-Pendergrass.)

it difficult to keep the foreign bodies in the bronchi of their animals long enough to permit their students to remove them through the bronchoscope. Coryllos and Birnbaum found it necessary to fix their obstructing balloons to the walls of the bronchi by ingenious expanding metal prongs.

We further believe that with material of the proper viscosity atelectasis can be produced at will, without any alteration in the diaphragm other than that produced by the variation in the negative pressure of the pleural cavity which follows the atelectasis.

In our first experiments, we were careful to include all the suspected etiologic factors. The dogs were narcotized with morphine, anesthetized with ether and an operative incision was made through the

Fig. 4 (dog 456).—Three hours after introducing into main bronchus of the right lung the obstructing bronchial secretion taken from main bronchus of the left lung of C. M., a patient with postoperative massive atelectasis (figs. 1 and 2). (From the Department of Surgical Research, University of Pennsylvania; Lee-Tucker-Clerf-Ravdin-Pendergrass.)

upper half of the right rectus muscle, entering the abdominal cavity. This wound was closed with layer sutures and then strapped with broad adhesive plaster which encircled the lower portion of the costal arches, as we dress so many upper abdominal wounds. Finally the dog was laid on his right side, the so-called Sims position commonly used in the

postoperative treatment of appendiceal abscess and peritonitis. Sodium iso-amyl ethyl barbiturate was then injected intraperitoneally, the amount varying from 25 to 50 mg. per kilograms of body weight. This resulted in profound anesthesia, with abolition of the cough reflex lasting from five to seven hours. With the dog lying on his right side, the bronchoscope was introduced into the main bronchus of the right lung, and 6 cc. of the secretion previously removed by Dr. Clerf from the patient at the

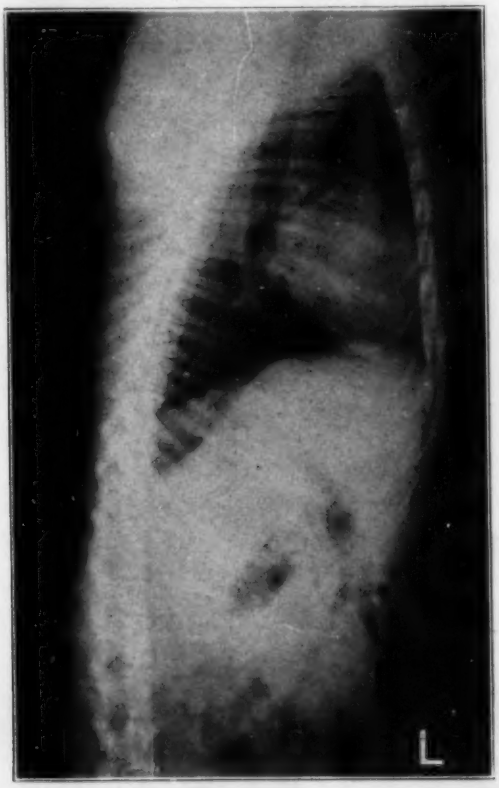

Fig. 5.—Twenty-four hours before experimental production of massive atelectasis. (From the Department of Surgical Research, University of Pennsylvania; Lee-Tucker-Ravdin-Pendergrass.)

Pennsylvania Hospital, or, subsequently, the synthetic substance of acacia, was introduced and bronchial obstruction produced.

With the loss of the cough reflex, respiratory efforts become deeper and the entire mass of bronchial secretion or acacia is drawn further into the main bronchus. A few minutes after the completion of the introduction of the bronchial secretion or synthetic preparation, and following the

removal of the bronchoscope, definite respiratory distress develops. This distress is often so marked that it seems for a time that the dogs are about to die. The respiratory movements finally become regular and rhythmic, although they are unusually slow, due probably to the sodium amytal. The movements of the chest on the obstructed side become restricted, while those of the opposite side are very much exaggerated, and there is a distinct bulging and a visible increase in the size of the thoracic cav-

Fig. 6 (dog 456).—Three hours after exploratory laparotomy and blocking of the main bronchus of the right lung with secretion removed from main bronchus of the left lung of C. M., patient with postoperative massive atelectasis (fig. 2). (From the Department of Surgical Research, University of Pennsylvania; Lee-Tucker-Ravdin-Pendergrass.)

ity on the unobstructed side. The apical impulse of the heart shifts to the affected side within three hours.

In the second group of experiments the operative procedures, the morphine narcosis and ether anesthesia were omitted, only sodium isoamyl ethyl barbiturate being given. Obstruction was produced by introducing a mixture of acacia into the main bronchus of the lobe in which

atelectasis was to be produced. We were just as successful in producing atelectasis by thus obstructing the bronchi after inhibiting the cough reflex with sodium iso-amyl ethyl barbiturate as we were when including the other operative factors of an abdominal incision, morphine narcosis and general anesthesia.

In the third group of cases, we added opaque substances to the viscid acacia mixture, and we were able to demonstrate radiographically by

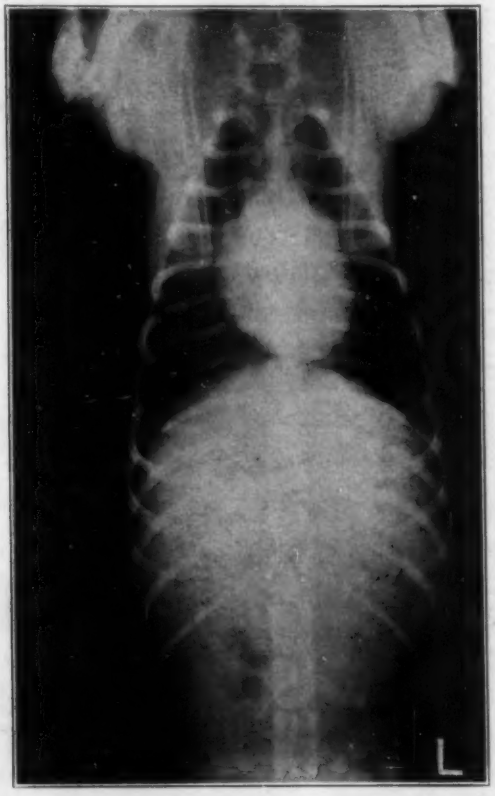

Fig. 7 (dog 555).—Twenty-four hours before experimental production of atelectasis. (From the Department of Surgical Research, University of Pennsylvania; Lee-Tucker-Ravdin-Pendergrass.)

the use of bismuth or iodine suspended in acacia the obstructing material in the bronchus leading to the atelectatic tissues.

In conclusion, we wish to reiterate our belief that from an experimental standpoint, the crux of this work lies in our ability to abolish the cough reflex. When we first introduced the bronchial secretion obtained from

man into the bronchus of our dog, in spite of the preoperative morphine narcosis and the efforts of Dr. Ravdin to produce deep ether anesthesia, the dogs' efforts to expel the secretion were so persistent and successful that we despaired of producing obstruction. Dr. Ravdin then introduced intraperitoneally 250 Gm. of sodium iso-amyl ethyl barbiturate and within a few minutes the narcosis was so deep that the cough reflex was absolutely abolished and remained so for from three to four hours. The

Fig. 8 (dog 555).—Three hours after the introduction of acacia into the main bronchus of the right lung; massive atelectasis and transposition of the heart to the right. (From the Department of Surgical Research, University of Pennsylvania; Lee-Tucker-Ravdin-Pendergrass.)

impossibility of producing bronchial obstruction in dogs unless the cough reflex is abolished has been experienced over and over again, and we feel that this evidence is of peculiar significance, both experimentally and clinically.

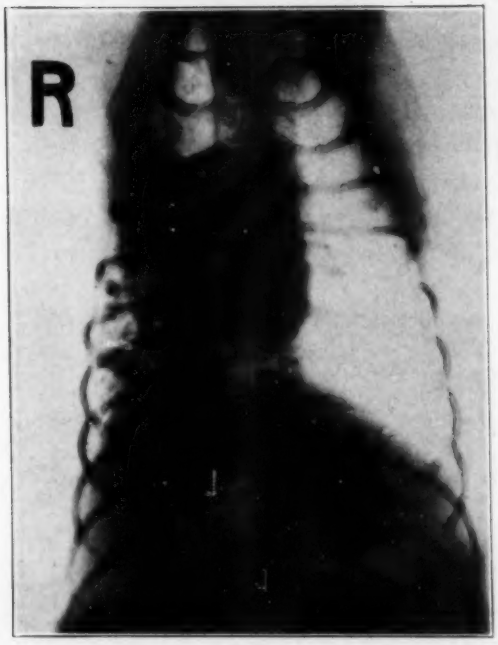

Fig. 9.—Fifty minutes after the introduction of an opaque acacia mixture into the right main bronchus of a dog.

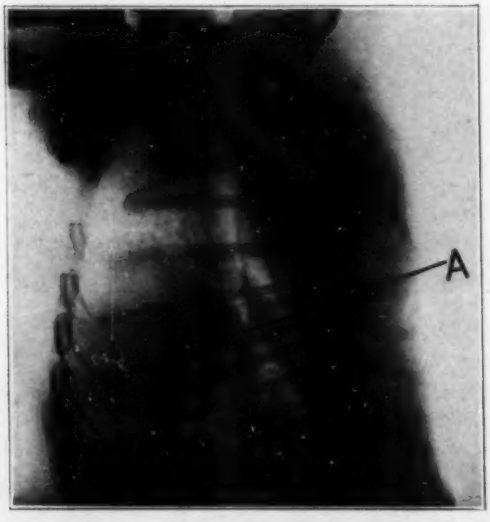

Fig. 10.—Lateral view taken three hours after the introduction of an opaque acacia mixture into the right main bronchus of a dog; obstructed bronchus *A* and massive atelectasis are noted.

ABSTRACT OF DISCUSSION

ON PAPERS BY DRS. CORYLLOS AND BIRNBAUM, AND LEE, TUCKER AND PENDERGRASS

DR. I. S. RAVDIN, Philadelphia: As Dr. Lee and Dr. Coryllos have both shown, the crux of the problem experimentally is the maintenance of either the rubber balloon or the synthetic material, such as we have used, in the bronchial tree. We have found, as has Dr. Coryllos, that sodium amyl-ethyl barbiturate was the anesthetic of choice in the conduction of this work. It assists in abolishing the cough reflex over a period of hours.

We have studied chemically the material from cases of atelectasis, from cases in which bronchoscopic drainage has been practiced for bronchial asthma and from cases of lobar pneumonia. As yet we have found nothing striking in this work. The viscosity of the material removed by bronchroscopic drainage has been estimated and in the synthetic preparation we have the material approximately of the same viscosity. It is our opinion that the production of atelectasis is purely mechanical, and that there is no chemical feature in the material which assists in its production.

Recently, in the Laboratory of Surgical Research, we have been using an opaque material so that we could visualize the site of obstruction.

In going over the x-ray pictures of a number of cases of apparent postoperative pneumonia, we have satisfied ourselves that lobular atelectasis does occur, and that in many cases it is the precursor of lobar atelectasis. It is our opinion that if pictures are taken early enough, lobular atelectasis would be demonstrated in the majority of cases.

DR. WILLY MEYER, New York: When Dr. Scrimger of Montreal recalled to the minds of American medical men several years ago what he called at that time massive collapse, I think he did a great service, not only to the American medical fraternity, but to suffering humanity in general. It had not been discussed thoroughly here, so far as I know. At first we could not understand it under the name of massive collapse. A collapse of the lung in a closed chest, seemed, after all the studies made in the last twenty-five to thirty years, hard to comprehend. So I think it is of importance that the speakers of this morning have substituted the term that was used in the middle of the last century for this trouble: atelectasis.

We understand that plugging of the bronchus causes gradual absorption of air in the alveoli. The lung shrinks, with the natural consequence that in the normal thorax reduction of space must be made up. It is made up by the rising of the diaphragm on the affected side and by the healthy lung pushing the contents of the mediastinum over to the other side. With this in mind, I think that the picture has been cleared greatly, and we must be grateful to the two speakers and their associates for having gone to this extent into the real meaning of the pathologic picture, finding and showing what was surmised many years ago. But then there was no bronchoscope. Now it is found that this collapse of the lung is caused, not by a foreign body that enters from outside, but by a foreign body made by nature—a thick, viscid fluid which usually cannot be coughed up by the patient, not even by means of forced external manipulation, but can be aspirated from outside with the help of the bronchoscope. What can be more natural in the face of present developments than that the bronchoscope is introduced into the bronchial

tree, and that the viscid mass which is plugging the bronchus is sucked out? The rest is coughed out, and in a few hours everything is restored to normal. It is a wonderful advance and truly inspiring.

These facts having been established, it was but one step forward to say and to try to prove, by animal experimentation, that lobar pneumonia is a "pneumococcic," a bacterial massive atelectasis of the lung. To my mind, that again has brought on enormous advance. It does at last bring to fruition what many of us have been wishing for years, that the internist is now following in the footsteps of the surgeon in joining hands with the bronchoscopist and considering him his best friend in the joint treatment of patients with conditions of the lung.

In the treatment of a disease which still takes the life of so many people, since it has been found that also in lobar pneumonia the bronchus is plugged primarily by a mass that can be removed by aspiration, irrespective of high fever, the internist has begun to call in the bronchoscopist! It stands to reason that if the bronchus in a patient affected with acute pneumonitis has been successfully cleared, the air can reenter the bronchus. We know that oxygen has a healing influence on inflammatory conditions. And, furthermore, with the bronchus cleared there is better chance for internal, immediate drainage. Although we are just in the beginning of this scientific therapeutic cooperation, the first step at least has been made, and we cannot rejoice too much.

DR. HOWARD LILIENTHAL, New York: From what has been seen today I do not see how anybody can fail to have been convinced that there is such a thing as obstructive, massive atelectasis.

I do not believe, however, in spite of this work and its results, that the last word has been said in the matter. In the first place, I am almost convinced that massive atelectasis, although it occurs or may occur on account of obstructive conditions, may also occur for other reasons, one of these is a probable spastic condition of the lung. There is not time for me to go into the details here. Only a short time ago I had the opportunity to observe the actual production of the condition of massive collapse. I was operating at Bellevue Hospital on a man who had a fibroma as large as a grapefruit, attached to the posterior upper part of the thorax. During the operation, which was done under intrapharyngeal pressure, there was a sudden collapse of the right, upper pulmonary lobe, the other two lobes remaining exactly as they were. The man died within forty-eight hours, and on postmortem examination, the lobe had not yet expanded. There was no possible reason to think that there was any obstruction in the bronchus of this patient, and there certainly was none at the time of the postmortem section. Dr. Morris who was assisting me in the operation, also saw the collapse which occurred within a few seconds. In other cases which have been reported, notably an important one of Dr. Bergamini, a laparectomy had been performed, and as the last stitch was tied, death occurred. At the postmortem examination massive collapse of both lungs was found and no obstruction of the bronchi. There had not been time for air to have been absorbed in the normal way out of the alveoli, and yet the lung, except in its upper part, on each side, was completely atelectatic and liver-like. The diaphragm was drawn up to the fourth rib anteriorly. I got these details from Dr. Douglas Symmers, who was responsible for the autopsy.

Interesting as these experiments are, I believe that a great deal more work must be done in order to show that, lobar pneumonia is a purely obstructive

disease. In the first place, on x-ray examination we do not find great displacement of the heart in lobar pneumonia. That is an important point. We do not find the characteristic appearance which is present in massive collapse or massive atelectasis, whichever you choose to call it. There we do find striking displacements of the mediastinal structures. In lobar pneumonia we do not find them. Also, lobar pneumonia usually begins with a chill, even before physical signs indicate the presence of an actual solidification. Not only that, but with the crisis there is no change in the solidity of the lung; that comes later, although the temperature may have dropped suddenly from 106 F. to normal, and the patient's sense of well-being may have become established. These are some of the phenomena which must be explained.

Besides, there are certain chemical examinations, for example, the comparison of the chloride content of the urine in cases of pneumonia with that in cases of massive collapse; also, as has been suggested here, the comparison of negative pressure within the thorax in both cases, and that, I am sorry to say, cannot be done very well in the case of human beings.

I do not wish, under any circumstances, to belittle or criticize unfavorably any of the work which has been presented here. In fact, I think that when there is a massive collapse after an operation I would be the first one to call in the bronchoscopist to help me. It could do little harm, if any, and it might do considerable good in case there was an obstruction which could not be expelled by ordinary coughing. I only say that we must not consider that either the problem of pneumonia or that of massive collapse has been solved by what we have heard here today.

Dr. F. T. Lord, Boston: In my experience in 558 patients with pneumonia, personally examined, there was a complicating collapse of the lung embracing a sufficiently large area to lead to clinically recognizable cardiac displacement in fourteen or 2.5 per cent. The complication is more frequent in children than in adults probably because of the greater readiness with which small bronchi become plugged by exudate. As the collapse of only relatively large areas leads to clinically recognizable cardiac displacement, the collapse of small areas may well be much more frequent than my figures indicate. The recognition that collapse occurs in the course of pneumonia is of practical importance because of the readiness with which it may be confused with pleural effusion. In some instances, there has been with the pneumonia a collapse of the lung and a pleural effusion on the same side. The condition is of importance also because of the danger of trapping infectious material beyond the bronchial occlusion. In certain cases the collapse has occurred in patients who have been allowed to lie constantly on one side, and it therefore seems desirable that patients with pneumonia be turned from the back to one or the other side frequently during the course of the disease in the hope that by this means the complication may be avoided.

Dr. J. J. Singer, St. Louis: In the last week Dr. Graham and I came across a roentgenogram of a patient who had been under pneumothorax treatment for thirteen months. She had tuberculosis of the right lung with immense cavities in the apex and in the base. Now the question of massive collapse, as brought out by the essayists, has led me to an entirely new train of thought, and this picture is some evidence of how quickly one grasps at an idea that one has not thought of before.

In figure 1, the heart is in the right side of the chest. There is pneumothorax and the upper lobe is attempting to expand while the lower atelectatic lobe shows the bronchi collapsed. In the lower lobe of the right lung there was originally a large cavity. When the patient expired deeply (fig. 2), the heart would shift to the opposite side, approximately to its normal position. The dextrocardia, I think, is due to an increased pleural negative pressure secondary to the fixation of the lower lobe, in a state of atelectasis. We introduced a little air, thus reducing the negative pressure in the right pleural cavity, and immediately the heart and atelectatic lung moved over into the opposite side of the chest.

In picking out patients with unilateral involvement for thoracoplasty, we often see in the picture marked density involving the whole lung, with displacement of the heart. The dense shadow we interpreted as fibrosis. I am now wondering,

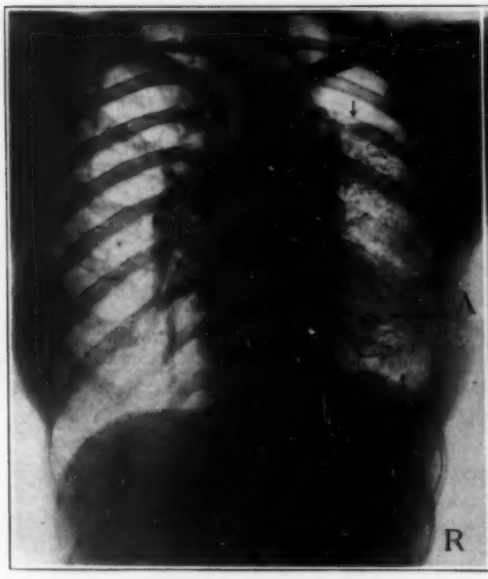

Figure 1

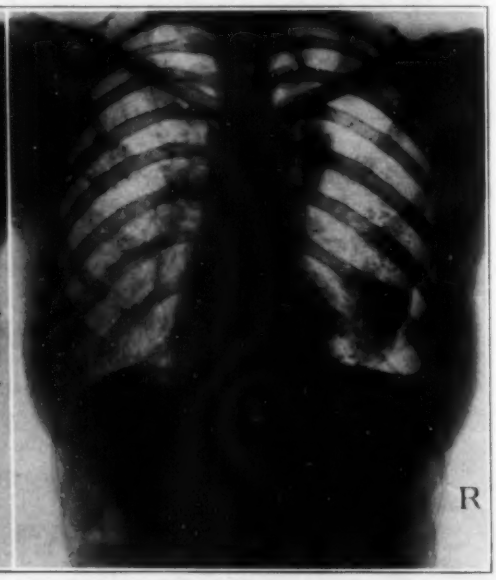

Figure 2

The figure on the left shows A. M., on April 20, 1928, when iodized oil 40 per cent was introduced into the right lung. Pneumothorax and compression of the lower lobe of the right lung is evident. This roentgenogram shows the heart pulled over into the right side of the chest because of atelectasis of the lower lobe. The pneumothorax is outlined by the arrows and the atelectatic lung is indicated by *A*. The figure on the right was taken April 23, on expiration. The heart is shown in the left side of the chest in an almost normal position. The roentgenogram shows evidence of remaining iodized oil.

and I would like some further discussion on the topic, whether or not the dense shadow represents in reality massive collapse, which has no relationship to fibrosis but which may be due to plugged bronchi or to increased negative pleural pressure.

Dr. N. W. Green, New York: All or many of us are familiar with Dr. Francis Delafield's "Pathology of the Lungs" in Delafield and Prudden's "Pathology." The conclusions in this textbook were arrived at by the methods then

at hand, namely, by autopsy on the dead subject, but now we have other means of collecting material for a new "Pathology of the Lungs." Dr. Lee and Dr. Coryllos are showing us the way by the newer instruments, that is, by the use of the bronchoscope and by the concomitant use of the x-ray pictures.

DR. ALEXANDER LAMBERT, New York: I was much interested in the work with Dr. Coryllos because it demonstrated new facts and it also showed distinctly the effect of bronchoscopy on patients with pneumonia. It did not injure in any way the seven who had bronchoscopy practiced on them. Several said that they felt it was rough but that it made them feel better. I think it is always well to take the patient's point of view as to whether a new treatment is successful or not.

Most of these men were alcoholic addicts and most of them were young; they withstood the effects of bronchoscopy well, but not of pneumonia. As far as bronchoscopy on the course of the pneumonia was concerned, in one elderly man it showed a definite temporary striking result. His temperature was 106 F.; it dropped down within twenty-four hours to normal and stayed there for twenty-four hours; it stayed around normal, under 100 F. Then it slowly rose again, and he died from a general pneumococcic infection with an extension of pneumonia.

We had a younger man, also an alcoholic addict. He showed a beginning pneumonia, and by removing the plug from the bronchus the physical signs of tubular breathing changed to those of rough breathing; the condition ran a short course with definite benefit, and the patient rapidly recovered.

In the majority of the patients bronchoscopy seemed to give more a sense of physical relief and a feeling of improvement than an effect on the actual course of the pneumonia. It did not seem to affect the course of the pneumonia one way or another. It definitely affected the physical signs. The physical signs, when the plug was obtainable and could be taken out of the bronchus, changed from definite tubular breathing to harsh breathing; the pneumonia was of the lobulated type, in patches, while previously it had been tending toward the lobar type.

So far it is a proceeding, as far as I can see, that may be of advantage. It certainly does not do any harm. The pathologic process that has been shown in the roentgenograms of the movement of the heart to the side of the affected lung in the early stages of pneumonia is interesting. By percussion there is a demonstrable variation of as much as 1 or 1½ inches in the movement of the heart from one side to the other. I did not believe that one or two of the patients had pneumonia. The heart was over to one side as much as 1 or 1½ inches, to the right side particularly, and was demonstrably so by percussion. I took them into the fluoroscope room and found pneumonia present in the lung on that side, which convinced me that there was a definite change in position of the heart of a human being in the early stages of pneumonia, that could be demonstrated. Yet that is new to me, and I cannot say how often it occurs. It has been demonstrable in probably five of the seven cases we had.

DR. POL N. CORYLLOS, New York: I shall answer Dr. Lilienthal with regard to atelectasis of the lung. He cites the case of Bergamini and Shepard and his own case in which "collapse" of the lung was produced instantaneously during the operation. We know from experimental evidence that it is possible for the absorption of carbon dioxide and oxygen to take place in a few minutes, but that absorption of nitrogen requires twenty hours or more. Apparently

we cannot explain by the theory of obstruction how atelectasis can be produced so rapidly. But if oxygen and carbon dioxide is absorbed in a few minutes, it yet remains to be considered how quickly a mixture of ether and air in the lungs of the patient operated on will be absorbed. In experiments on dogs, if one administers a mixture of ether with air in about the same proportion as is given to patients, and then plugs the bronchus, the ether fraction is absorbed almost instantaneously, much more rapidly than the carbon dioxide; this may explain those cases of rapid production of massive atelectasis that have been reported.

Furthermore, I do not believe that spastic contraction of the bronchial muscles can produce atelectasis, because in every one of those cases in which spastic contraction of the bronchial muscles obtains — as in asthma or in anaphylactic shock in the guinea-pig — atelectasis has not been observed.

Failure of some workers to produce experimentally typical atelectasis of the lung would indicate that a complete obstruction of the bronchus was not affected. With regard to the bronchoscopic treatment of patients with pneumonia, there are many handicaps. The main one is that whereas in atelectasis there is no pathologic condition below the obstructing "plug," in pneumonia there are inflammation and rapid exudation into the alveoli. Herein lies the great difference between the two diseases. That is why there is a great displacement of the heart in one instance and slight or none in the other. For this reason, we insisted in our paper that in pneumonia bronchoscopic treatment must be given early. Furthermore, in the future—and the experimental evidence is encouraging—we may perhaps help nature bring about an early dissolution or liquefaction of the consolidated mass so that its absorption or expulsion will occur more rapidly.

MASSIVE ATELECTASIS COMPLICATING PARAVERTEBRAL THORACOPLASTY FOR PULMONARY TUBERCULOSIS *

FRANK B. BERRY, M.D.
NEW YORK

Ever since the application of surgical measures to pulmonary tuberculosis with the final development of the paravertebral thoracoplastic operation, one of the more frequent and dreaded complications in the immediate postoperative period has been pneumonia. This seems to occur either in the lung of the side on which operation has been performed or in the contralateral lung with almost equal frequency. Furthermore, there is a general unanimity of opinion that this pneumonia, generally tuberculous, is caused by aspiration of infective material from the diseased area. Following thoracoplasty, such aspiration is unquestionably favored by the weakened wall of the chest and the pain incident to the operation, both of which interfere considerably with the ease, mechanism and effectiveness of cough and expectoration —vital factors in keeping a tuberculous lung rid of its noxious waste. Various methods of procedure have been recommended, and scrupulously adhered to, in order to prevent the occurrence of this unfortunate complication or at least to lower its incidence. The majority of surgeons have followed the method of Sauerbruch and remove the lower ribs first, thereby compressing the lower lobe so that its chances of infection by aspiration are greatly diminished.

Lambert and Miller, however, reverse the process. They have felt that the initial removal of the lower ribs interferes with cough and expectoration; hence that it is wiser to confine the first stage of the operation to the upper ribs. With the resulting compression of the upper lobe, the patient is thus able to cough up his secretions with maximum efficiency and comfort.

Alexander [1] has suggested a still further improvement on theoretically logical grounds—a phrenicotomy as the initial procedure. This decreases the size of the lower lobe almost as much as thoracoplasty of the lower ribs and actually favors cough. A few weeks later, the upper ribs are resected. The lower part of the chest can be collapsed at a still later date should it be necessary.

Another method is the institution of a preliminary artificial pneumothorax whenever possible, this to be partly withdrawn following

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* From the Tuberculosis and First Surgical (Columbia) Divisions, Bellevue Hospital, clinic of Drs. J. A. Miller and A. V. S. Lambert.

1. Alexander, J.: Surgical Treatment of Pulmonary Tuberculosis.

operation if the condition of the patient demands it. By this regimen, the lower lobe is collapsed and aspiration prevented following initial removal of the upper ribs. There is no interference with the cough mechanism, and there is apt to be less mediastinal shift than when pneumothorax has not been previously employed.

Yet pneumonia still persists with higher morbidity and mortality than as a complication of other surgical procedures. Is this pneumonia always a true pneumonia? My experience leads me to believe that it is not. For this reason I wish to present four cases, evenly distributed over four years, in which the condition would ordinarily have been classed as postoperative pneumonia. Three are instances of proved massive atelectasis, two in the contralateral lung, and the fourth case is placed in this group on strong supportive clinical evidence. Both of the contralateral cases were fatal; in both of the homolateral ones, the patients recovered without developing pneumonia.

REPORT OF CASES

CASE 1.—M. H., a girl, aged 20, had been under treatment in Bellevue Hospital in the tuberculosis outpatient department and wards for chronic pulmonary tuberculosis for two years and had not quite held her ground. During this time an appendectomy had been performed for tuberculosis of the appendix, from which she made an uneventful recovery. Roentgenograms made during this period showed a productive type of lesion in the left lung, most marked in the upper lobe, where there were several small cavities. There was also fibrosis and retraction of the heart and mediastinum to the left. There was, in addition, slight fibrosis of the apex of the right lung.

On Oct. 6, 1923, she was readmitted to Bellevue because of hemoptysis. Artificial pneumothorax seemed to control this, but had to be repeated every three or four days and caused pain and temperature reaction. On admission, her temperature was 98 F.; the pulse rate, 88, and respiration, 20. Early in November, however, the temperature varied from 98 to 101.4 F. and the pulse rate from 80 to 134, with an average level of from about 100 to 110. The blood count showed: white blood cells, 7,400; polymorphonuclears, 65 per cent; and red blood cells, 4,100,000, with hemoglobin, 70 per cent. Roentgen examination at this time showed a localized area of infiltration involving the lower half of the upper lobe of the right lung. There was a similar lesion of greater extent involving the upper lobe of the left lung, evidence of a small collection of air in the left pleural cavity and small cavity formations in the upper lobe.

A diagnosis of tuberculosis and pneumothorax of the left lung was made.

In view of the ineffectuality of pneumothorax and the reactions it caused, it was decided to perform a thoracoplasty. The lesion in the right lung seemed to be inactive, whereas over the left lung, just before the start of pneumothorax, extensive dulness, râles and bronchial breathing were noted.

On December 14, a paravertebral thoracoplasty was performed with partial removal of the upper ten ribs. On December 15, twenty-four hours after operation, the patient was dyspneic and cyanotic. There was no change in percussion, but the breath sounds were distant. The temperature was 101.8 F.; pulse rate, 140, and respiration, 48. The next day there was marked paradoxic breathing, and a few fine râles were heard on inspiration. On December 17, dulness was noted over

the entire right side of the chest, many fine râles were heard and there was harsh breathing in the axilla. A diagnosis of pneumonia was made. The condition gradually became worse, and the patient died on the morning of December 19, six days after operation.

At autopsy, the heart was found displaced to the right midclavicular line. There was a pneumothorax with some fluid in the left pleural cavity and many firm adhesions between the visceral and parietal pleurae. The lung itself was completely collapsed, and on section was darkened with mottled caseous areas.

The right lung was collapsed to about one-third its normal size, and there were fresh adhesions present. The lung felt leathery and elastic with increased consistence. On section, the cut surface varied from a dull red to opaque yellow, and several small areas of tuberculous infiltration were found. The lung seemed almost airless. Unfortunately, the bronchi were not examined carefully. Pneumonia was not present.

The anatomic diagnoses were: tuberculosis of left and right lungs; pneumothorax, left; pleural effusion, left; fibrinous pleuritis, right; and massive collapse of the right lung.

Case 2.—John M., aged 26, was admitted to Bellevue Hospital on Oct. 1, 1927, from Loomis Sanatorium where he had been under treatment for pulmonary tuberculosis for one year. On physical examination, the right lung was found normal. The left side of the chest was dull to the level of the sixth spine posteriorly and the third rib anteriorly. The breath sounds were somewhat distant and bronchovesicular. There were medium moist râles most numerous at the hilum. The temperature was 98.8 F.; pulse rate, from 78 to 110, respiration, 26.

Roentgen examination showed: irregular consolidation of the base of the left lung with a large empty cavity in the lower portion of the upper lobe. There were peribronchial fibrotic changes in the right lung. The heart was slightly retracted to the left.

Notwithstanding what appeared to be extensive pleural adhesions, artificial pneumothorax was attempted on October 7, and 200 cc. of air were injected. This resulted in partial collapse of the upper lobe and its cavity. Again on October 17, 350 cc. of air were injected.

On October 25, a first stage paravertebral thoracoplasty was performed with removal of the upper six ribs. The patient stood the operation well and was returned to the ward in fair condition with a pulse rate of 140; the rate soon dropped to 120. That afternoon he suddenly became cyanotic; his pulse rate rose suddenly to 160; he was covered with a cold perspiration, and complained of inability to breathe. Owing to pain, he had maintained up to this time a shallow respiration. He was urged to breathe more deeply, and he coughed up a small amount of thick mucus. As his systolic blood pressure was only 95 mm. of mercury and his condition seemed to be critical, he was given 1 ampule of strophanthin intravenously and a transfusion of 500 cc. of whole blood. Following this, his condition was much improved.

On October 26, in the morning, his temperature was 101 F.; pulse rate, 116, and respiration, 24. Respiration, however, was still very shallow and almost entirely abdominal. That evening he was coughing up thick mucus; there was again cyanosis of his lips, and there were coarse and fine râles at the base of the right lung. The temperature was 102.8 F; pulse rate 140, and respiration, 32. He was urged to breathe deeply and was made to cough, but without improvement. An x-ray picture taken later in the evening showed the right side of the diaphragm elevated, the heart and mediastinum displaced to the right and a diffuse haziness

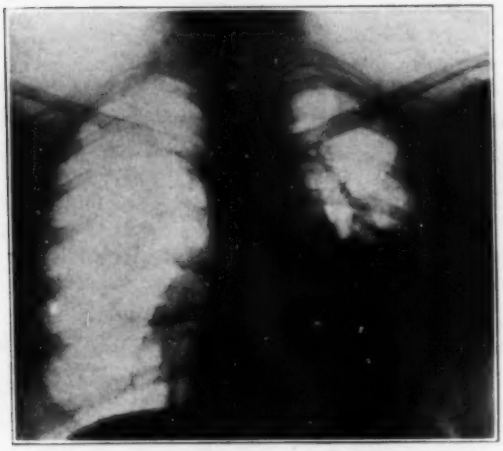

Fig. 1 (case 2).—Before operation; lesion on the left.

Fig. 2 (case 2).—Postoperative roentgenogram taken just before death showing shifting of the heart and mediastinum to the right, clouding of the base of the right lung and narrowing of the intercostal spaces.

at the base of the right lung. The diagnosis of massive atelectasis was made, but he was thought to be too ill to permit bronchoscopic treatment. This was undoubtedly an error in judgment. His condition rapidly became worse and he died on the morning of October 27.

At autopsy, many adhesions were found in the left pleural cavity. The lung was collapsed and contained many conglomerate caseous tubercles. No cavities were found. The right lung was small and firm and seemed greatly congested. There was still slight crepitation, and portions floated in water. Thick mucous plugs were expressed from the tertiary bronchi in the lower lobe. The upper lobe contained a few isolated tubercles. Thus an extensive though partial collapse of the right lung was found with the mucus in some of the bronchi.

CASE 3.—E. L., a woman, aged 22, had been under treatment for chronic pulmonary tuberculosis of the right lung for several years. There was a large cavity ocupying almost the whole of the upper lobe, and there was also a moderate involvement of the middle and lower lobes. On Jan. 3, 1925, a right phrenicotomy was performed which resulted in improvement in the patient's condition. On April 17, a first stage paravertebral thoracoplasty was performed with removal of the upper five ribs. Following operation, the patient was in a state of considerable shock; breathing was very shallow, and there was inability to cough, although she complained constantly of the thick mucus she wanted to raise. With urging and constant assistance, she was able at times to cough up some of this. The next day she was very uncomfortable with dyspnea and cyanosis, and was bathed in cold perspiration. The pulse rate was 160. At this time her condition was extremely grave, and the possibility of a massive collapse was considered, as there were dulness and absence of breath sounds over the lower lobe. An x-ray picture taken at this time confirmed the diagnosis and showed the heart and mediastinum retracted well over to the right and cloudiness over the lower and middle lobes of the right lung, with collapse of the upper lobe from operation. Shortly after this the cough became more productive, she began to improve and eventually made a complete recovery without the development of pneumonia. Although the collapse over her cavity seemed adequate at first, some cough and sputum persisted. Fourteen months later, therefore, the collapse was completed by removal of the lower ribs. Now, two years later, she is well.

CASE 4.—J. M., a man, aged 48, was admitted to Bellevue Hospital on Oct. 3, 1927, from Loomis Sanatorium, where he had been under treatment for pulmonary tuberculosis for the past fourteen months. During the first two months of his illness he had had several artificial pneumothorax treatments; these had been discontinued, however, because of adhesions. In spite of these treatments, there were repeated hemoptyses. While at Loomis Sanatorium, he had been confined to bed most of the time because of the hemoptyses. Physical examination of his lungs showed dulness over the upper lobe of the left lung to the second rib anteriorly and the third spine posteriorly and also at the extreme base posteriorly and in the axilla, where the note was almost flat. Breath sounds were everywhere distant, and there were numerous pleural and medium râles over the entire chest. Fremitus was increased except at the base and in the axilla, where it was diminished. The right lung was normal. The temperature varied from 98 to 99 F.; the pulse rate from 90 to 100, and the respiration was 22.

Roentgen examination showed: infiltration, fibrosis and cavitation in the upper lobe of the left lung and a small patch of consolidation at the base of the same lung.

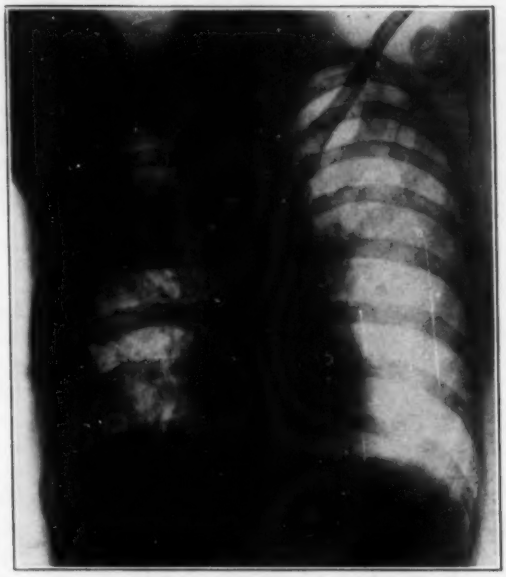

Fig. 3 (case 3).—Before operation; lesion on the right.

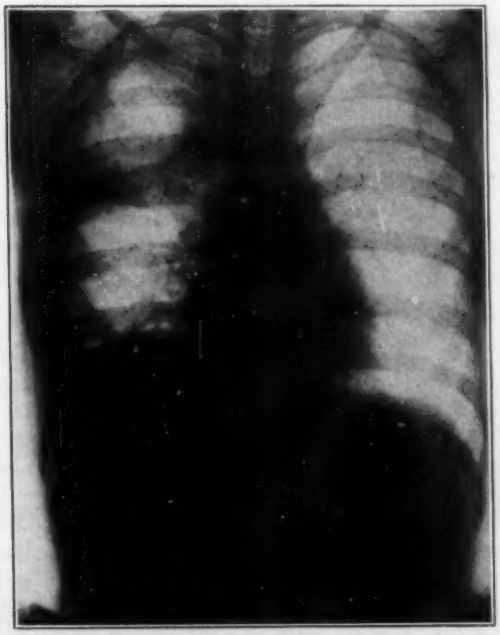

Fig. 4 (case 3).—After phrenic neurectomy.

Fig. 5 (case 3).—Postoperative atelectasis of the lower lobe of the right lung. The heart is retracted to the right.

Fig. 6 (case 3).—Postoperative roentgenogram, after the disappearance of atelectasis.

On October 14, first stage thoracoplasty was performed, with removal of the upper five ribs. At the end of the operation, the patient's condition was fair. The pulse rate was 134; the blood pressure was 90 systolic, 20 diastolic; four hours later it had risen to 100 systolic and 78 diastolic. As there seemed to be considerable oozing and his blood pressure dropped to 80 systolic and 60 diastolic, the pulse rate remaining the same, he was given a transfusion of 500 cc. of whole blood sixteen hours after operation with immediate rise of blood pressure to 118 systolic and 80 diastolic; the pulse rate remained the same, 132, and the temperature, 101.6 F.

Four hours later, twenty hours after operation, his temperature suddenly jumped to 103.6 F.; he was covered with clammy perspiration; his pulse rate was 160 and thready, and he was somewhat cyanotic. Coarse moist râles were heard over the lower lobe of the left lung. Nothing else was noted at the time, and the house-officer considered the condition to be a beginning bronchopneumonia.

No x-ray picture was taken. Shortly after this, the patient began to cough up some rather thick mucous and seemed to feel easier. Three and a half hours later his pulse rate had dropped to 120 and the temperature to 101.2 F.; perspiration had ceased, cyanosis had cleared and examination of his lungs gave negative results. Following this he made a slow recovery, during which he developed a superficial infection of his wound and osteomyelitis of the greater trochanter of one femur.

On Jan. 12, 1928, three months after operation, he returned to Loomis Sanatorium for a further treatment.

In spite of the absence of roentgen evidence, the picture seems characteristic of the sudden development of a massive atelectasis in the lower lobe of the lung on the side on which operation had been performed; this cleared up with characteristic rapidity following relief by cough and unplugging of the bronchi of that lobe. I therefore believe that this case should belong in this series.

COMMENT

It is unnecessary here to dwell on the history and details of this extremely interesting condition. Within the past year, two excellent articles have appeared reviewing the entire subject. Each supplements the other. From my own clinical observations, I am wholly in accord with the conclusions of Coryllos [2] that massive atelectasis of the lung is always due to complete obstruction of a bronchus. Certainly in the foregoing cases this seemed to be the precipitating cause. In each case there was cavitation, and following operation great difficulty was experienced by the patients in getting rid of the thick tenacious sputum, which, as they said, seemed to strangle them.

In case 1, a complete single-stage thoracoplasty had been performed, which left the patient devoid of all support on that side for effective and expulsive cough. The true condition was unrecognized during life but was considered rather as an extensive bronchopneumonia. Unfortunately, the condition of the bronchi was not carefully noted at autopsy.

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2. Coryllos, P. N., and Birnbaum, G. L.: Obstructive Massive Atelectasis of the Lung, Arch. Surg. **16**:501 (Feb.) 1928.

Case 2 presented a similar picture. In case 1, artificial pneumothorax had been administered therapeutically several times prior to operation and had failed. In case 2, two treatments were given prior to the removal of the upper ribs with the hope of lessening the chance of aspiration. This was successful enough as far as the homolateral lung was concerned, but here again the atelectasis was contralateral. The condition was recognized, but the treatment was ineffectual. Bronchoscopy was not immediately available; moreover, the patient's condition seemed so desperate that at the time it was doubtful whether he could stand the instrumentation. Manual manipulation, however, and forced respiration were unavailing. At autopsy, the plugged tertiary bronchi adequately explained his atelectasis and emphasized what might have been accomplished by prompt bronchoscopic treatment.

Case 3 presents a different picture. An initial phrenicotomy had been performed some weeks prior to thoracoplasty to test the opposite lung, to provide some degree of rest to the diseased lung, and also to diminish the risk of aspiration in that lung. At the first stage operation, the upper five ribs were resected. Notwithstanding the precautionary phrenicotomy and the unimpaired lower part of the chest, the patient was unable to expectorate her thick sputum from the large collapsed cavity, and an atelectasis of the homolateral lower lobe occurred. Eventually she cleared her bronchi herself and continued to complete recovery.

Case 4 is somewhat similar to case 3, though much less severe, and falls into the group of cases classified by Mastics[3] as transitory atelectasis. The patient had the characteristic hectic onset with few physical signs. He improved rapidly, and there was complete disappearance of these signs after he had coughed up some thick sputum.

In the clinics of Drs. J. A. Miller and A. V. S. Lambert, we have performed 130 paravertebral thoracoplastic operations on 93 patients. Of these, 23 have single-stage complete operations, and in 8 instances the lower ribs have been removed at the first operation. In the remaining 57 cases and 85 operations, the initial major procedure was a paravertebral resection of the upper four to seven ribs. Whenever possible, we have preferred to precede operation with a partial artificial pneumothorax, which, we feel, decreases the volume of the lung and the possibility of aspiration as well as lessening the danger of mediastinal shift. We have not practiced phrenicotomy as a routine preliminary preparation, but rather have held it in reserve for cases with some definite indication.

In addition to the four cases already cited, pneumonia has occurred five times in this series. Twice it was contralateral and followed single-

3. Mastics, E. A.; Spittler, F. A., and McNamee, E. P.: Postoperative Pulmonary Atelectasis, Arch. Surg. **15**:155 (Aug.) 1927.

stage complete thoracoplasties, one of which, however, was a revision of a former operation. In the other three instances it was homolateral, following the removal of the upper ribs on that side, and in one of these cases a phrenicotomy had previously been performed. Thus the percentage of pneumonia has been 3.8 per cent for the entire number of operations, and 6.9 per cent for total cases of early complications of the lung.

In comparison with others, to quote from Archibald,[4] I find that in his series of 188 operations in 88 cases, fatal pneumonia occurred after 1.6 per cent of the number of operations. Brünner, from Sauerbruch's Clinic, reported two series: one of 99 cases with 25 per cent mortality from pneumonia, and another of 117 cases with a 1.7 per cent mortality. In Bull's series of 92 cases, there was a 2 per cent mortality from pneumonia; and in that of Jacobaeus and Key of 50 cases, there was also a 2 per cent mortality. In all of the foregoing quoted cases, with few exceptions, the procedure was operation in two or more stages with the lower ribs resected first.

Again, quoting from Archibald, in the American and Canadian statistics of 270 cases, 20 deaths were reported in the first three days after operation. The cause was usually said to be cardiac failure with paradoxic respiration, or myocarditis. One of the most constant signs of a massive atelectasis is a paradoxic respiration. It would seem, therefore, that some of the early fatalities reported from other clinics as due to heart failure, mediastinal shift and shock may possibly have been caused by an unrecognized massive atelectasis. These patients are obviously in an ideal condition for the development of atelectasis, the recognition of which is not difficult if this possibility is constantly borne in mind, as it should be in all thoracic and abdominal operations. Once suspected, it may, of course, be readily verified by roentgenograms. When diagnosed, treatment by the bronchoscope should be instituted immediately; failing this, attempts should be made to cause the patient to cough, as by manual manipulations such as slapping or turning the patient, by the use of an emetic or by the passage of a stomach tube. If not relieved, the mortality rate is high; at least the chance of a subsequent pneumonia with its accompanying dangers is materially enhanced.

As to prevention, the question at once presents itself whether the lower or upper ribs should be resected first. From an analysis of our cases, it is apparent that the discrepancy between mortality rate of 3.8 per cent and the 1.6 per cent of Archibald is not great. To be

4. Archibald, E.: Transactions of National Tuberculosis Association, 21st Annual Meeting, 1925; Transactions of National Tuberculosis Association, 22nd Annual Meeting, 1926, Am. Rev. Tuberc. **15**:564 (May) 1927; Brown, A. L., and Archibald E.: Am. Rev. Tuberc. **16**:3 (Aug.) 1927.

sure, if one adopts the mortality rate of 6.9 per cent, which includes the deaths from massive atelectasis, the difference is more pronounced. The foreign series lack sufficient exactitude for satisfactory comparison. All of these patients have a thin margin of safety, and the possibility of their reserve being lowered by lack of hemostasis or the presence of infection must be considered as affording a suitable preparation for the development of pneumonia. With primary removal of the lower ribs, the patient is more comfortable and, as indicated by the foregoing data, the risks of aspiration and mediastinal shift would appear to be minimized. So far the evidence seems to favor this as the method of choice. In order finally to determine this question, however, additional series of cases should be carefully recorded, some carried out one way and some the other.

Preliminary artificial pneumothorax, I feel, is worth while whenever possible. Although sound on theoretical grounds, I do not believe that routine preliminary phrenicotomy as a practical procedure accomplishes as much as initial collapse of the lower lobe, for we have had one case each of homolateral pneumonia and massive atelectasis following thoracoplasty in which this initial technic had been carried out. In any event adequate relief from pain, encouragement to cough and proper support of the side on which operation was performed are important adjuncts in the prevention of postoperative respiratory complications—either atelectasis or pneumonia.

CONCLUSIONS

1. Four cases of massive atelectasis following paravertebral thoracoplasty for pulmonary tuberculosis have been presented. The mortality rate was 50 per cent.

2. In addition, in a series of 130 such operations on 93 patients, postoperative pneumonia has occurred in five.

3. The percentage incidence of pneumonia for the entire number of operations is 3.8 per cent, and for total early complication of the lungs, 6.9 per cent. Two of the cases of pneumonia and one of massive atelectasis occurred following the single-stage complete thoracoplasty. The others were subsequent to first stage operations in which the upper ribs were removed.

ABSTRACT OF DISCUSSION

DR. J. A. MILLER, New York: I am familiar with all of these cases. I think that from the medical point of view there are some things we have learned. In the first place, there is every reason to feel that atelectasis resulting from plugging of the bronchus is perhaps more apt to occur in this type of operation than in some other surgical procedures, because of the character and amount of the secretion. That this tuberculosis sputum should easily plug the bronchus is not, I think, surprising. Dr. Packard has shown that in chronic tuberculosis a com-

plete gradual atelectatic condition of the lung can occur from the pathologic changes of tuberculosis alone, and some of the cases that we have been accustomed to think of as chronic massive fibrosis of the lung and pleura with the shifting of the mediastinum over toward that side would probably come in this category. Dr. Packard has reported cases which demonstrate this.

One of the most interesting cases of this series was the first one, I think. This girl had lived for forty-eight hours, I think Dr. Berry said, and the diagnosis of pneumonia was made. We did not suspect massive atelectasis. I cannot help feeling that one of the contributing factors was the performance of the operation in one stage.

In connection with a diagnosis when an x-ray picture may not be available, I want to report to this society the interesting suggestion made by Dr. Habersman of Baltimore. He has had several cases of massive collapse following thoracoplasty in which he found that there was marked intrapleural pressure in the collapsed side, so marked that it went way up to 200 or 240 mm. of water. It seems to me that in his suggestion there is a valuable aid to diagnosis, that is, the measurement of the intrapleural pressure, particularly when one is in doubt as to what condition the shift of the mediastinum might be due. Then he also suggests, and shows very well in his reports, that if there is a massive collapse and if an artificial pneumothorax is performed at the time the needle is in place, the symptoms from shock caused by massive collapse can be relieved, and that also as the mediastinum is pushed back it seems to favor the removal of the bronchial plug. It seems to me that those suggestions are of considerable value.

In connection with the cases of Dr. Berry, I believe that massive collapse constitutes one of the complications of thoracoplasty against which one must always be on his guard.

Dr. Carl A. Hedblom, Chicago: The statement that prevention is better than cure has come to be more or less axiomatic in medicine. I think it applies to conditions such as pulmonary atelectasis occurring as a postoperative complication as discussed by Dr. Berry. Probably this complication develops chiefly because the patient's power to expel the sputum has been greatly reduced by an extensive thoracoplasty. If one does a divided operation, there is less interference with the cough reflex. I have never observed the complication in any of my patients. I am convinced that there is an increased factor of safety in the several-stage operation, and in case of doubt I would sooner do one stage too many than one too few.

Dr. Harold Brunn, San Francisco: In line with the last speaker's remark that prevention is worth many pounds of cure, I have one suggestion to make which I have put in practice following surgical operations for the past year and a half. It is my belief that those cases of so-called pneumonia, which develop suddenly the first day or two following operation with a high temperature and suddenly clear up by crisis in two or three days, are really atelectic in origin, involving small areas of the lung due to plugging of the terminal bronchus. It occurred to me, therefore, that to prevent the condition, especially after operation on the upper part of the abdomen, where the diaphragm becomes fixed and coughing is almost impossible and respiration shallow, the functions of respiration would be stimulated by giving carbon dioxide and oxygen. We have found that we can teach our nurses to do this. Three or four times for the first forty-eight hours carbon dioxide and oxygen is given for from three to five minutes. This produces deep respiration, usually followed by a coughing spell which clears the lungs. If it is persisted in more than five minutes patients are exhausted by the respiratory effort so that the condition of the patient is the criterion for the length of time it may be used.

Dr. Berry remarked that he attempted to make his patients take a deep breath and to cough during convalescence. We have also tried this method, but find that it is almost impossible to do this—many times on account of the pain and weakness of the patient. With the carbon dioxide and oxygen machine, however, bellows-like respirations are produced readily. I do not know, and have not tried, what effect this maneuver would have on cases of atelectasis of the massive type.

Dr. Evarts A. Graham, St. Louis: In line with what Dr. Brunn has been saying, I might say also that at St. Louis, ever since Henderson and Haggard proposed the use of carbon dioxide after ether anesthesia for the purpose of ventilating the lungs and getting rid of the ether more quickly by the deep respirations which are caused by the inhalations of carbon dioxide, we have been carrying out this procedure as a routine measure in all operations which are performed under general asesthesia regardless of what the general anesthetic has been. There is no question about the fact that carbon dioxide will make the patient take deep breaths. It will also frequently, as Dr. Brunn states, make the patient cough. I think it is of the utmost value, and I think that many of the other methods which are used to get rid of mucous plugs, etc., would probably be less necessary if inhalations of carbon dioxide were used more frequently.

Dr. John Alexander, Ann Arbor, Mich.: If it be true that acute atelectasis is caused by plugging of a bronchus with exceptionally tenacious mucus, it seems to me wise that we should consider what factor the giving of atropine before operation plays in the production of the mucous plug. Those of us who have personally taken as little as $\frac{1}{150}$ of a grain of atropine know how dry and tenacious the mucus of the nose and throat becomes. I believe, therefore, that we should give trial to omitting atropine as routine preoperative medication. An expectorant would be more logical, although scarcely to be recommended. In addition, it seems important that a patient with sputum should be given the advantage of gravity broncial drainage by keeping him at 20 or more degrees of the Trendelenburg position throughout the period of general narcosis.

Dr. Pol N. Coryllos, New York: There is no doubt that in the case of Dr. Berry the atelectasis was due to a "plugging" of the bronchus and "drawing" of the lung by aspiration of the material which was expelled from the affected lung. We can understand why in cases of this type he had a mortality rate of 50 per cent, and that the fatal cases were the ones in which a contralateral atelectasis later developed. There the good lung was obstructed, and the patient was not able to make up for the loss of the only lung which was useful to him. Such being the case, I consider that the best treatment would be to remove the cause of the obstruction by means of bronchoscopy, getting rid of the obstructing material by aspiration.

So far as diagnosis is concerned, I think that massive atelectasis is readily discovered provided one bears this condition in mind. I should also like to remark to Dr. Miller that the increase of negative pressure in the "collapsed" thoracic cavity exists but is not always detectable. The increase of negative pressure measures the resistance of the mediastinum. The negative pressure will be detectable only if the mediastinum is fixed.

I shall suggest another treatment in these conditions which may be preventive. In all cases of postoperative atelectasis of the lung the thing to do may be to perform bronchoscopy and to remove whatever can be aspirated from the other lung as well. There are no complications if the obstructing substance is expelled immediately. We know that even in severe cases bronchoscopy has not produced the least untoward effect on the patient.

Therefore, I am of the opinion that this is perhaps the best way of curing these patients or preventing this complication, the outcome of which is so uncertain. Bronchoscopy and aspiration may not get rid of the entire amount of mucus, but by creating a good airway the patient is subsequently able to expel enough of the obstructing material to keep the bronchi open.

Dr. J. J. Singer, St. Louis: In analyzing what I personally have learned at the thoracic surgery meeting this year I would say that the most important thing was the question of atelectasis as a new term in discussing so-called consolidations and changes in the lungs.

About four or five years ago when I first became interested in thoracic surgery, as far as this association was concerned, I brought the word "pneumonitis" to our hospital records, and our internists and our friends and associates did not know what I was talking about. They had heard of pneumonia, bronchial and lobular, but gradually they began to use the term "pneumonitis" as a definite pathologic condition. Now, in addition, we are using the word "atelectasis."

As we look back over the many cases that we have called abortive pneumonias and postoperative pneumonias, I believe we were dealing, as was well brought out by the essayist, with massive or partial atelectasis.

Dr. Berry: I do not want to leave the impression that we are advocates of the one-stage operation, because we are not. The operation in the first case was done very early in the series. We never do it in one stage now, but always in divided stages.

It seems to me that routine bronchoscopy is still a bit radical as a preventive measure; maybe not, but it seems so. We are heartily in agreement with Dr. Brunn as to the use of carbon dioxide, but unfortunately, at least in some of the New York municipal hospitals, such refinements have not yet met with much cooperation.

THORACIC TUMORS

GEORGE J. HEUER, M.D.
CINCINNATI

During the past two years I have been trying to assemble various groups of extrathoracic and intrathoracic tumors which have been reported in the literature. The incentive to do this was my agreement to write a chapter on thoracic tumors for new systems of surgery. In my early enthusiasm, the proposition did not seem so overwhelming, but as time has gone on, I wonder at my temerity in undertaking it. It soon became evident that certain tumors, for example the dermoid cysts of the mediastinum, are clearly defined pathologically, are well reported, as a rule, and do not offer any particular difficulties to one trying to assemble them. On the other hand, information concerning the pathology of other groups of tumors is in such a confused state that little of value results from their study. I have spent as long as three or four months on a single group of tumors in an effort to clear, in my own mind at least, the confusion which surrounds them, only to give up at last with a sense of the futility of the effort. As the symptomatology, diagnosis and intelligent treatment of intrathoracic tumors depends in a large measure on their pathology, I have been compelled to leave in an uncertain state the clinical manifestations and treatment of various groups of tumors. As a result of this confusion, there has come the desire for more definite information; it seemed to me that one method of securing this would be by establishing a thoracic tumor registry similar to that for bone tumors. A suggested plan for such a registry is to be presented by Dr. Andrus.

In presenting a report on the present status of knowledge concerning tumors, I shall not consider the tumors of the soft parts of the thoracic wall and the tumors of the clavicle and scapula, but only the tumors of the bony wall of the chest which may implicate the thorax, and the intrathoracic tumors.

TUMORS OF THE BONY THORACIC WALL

Reports concerning the tumors of the ribs and sternum have been fairly well compiled. In 1898, Parham collected ninety-seven cases and added two of his own. In 1913, Lund added twenty-nine cases, including one of his own. In 1921, Hedblom added thirty-five cases from the literature and forty-eight cases from the Mayo Clinic. Since 1921, I have found twenty-two cases in the literature and have five of my own. The total number of cases reported is about 240. Nearly 80 per cent of these were cases of tumors of the ribs and 20 per cent, tumors of the sternum. One hundred and seventy-seven (86 per cent) tumors were

primary; four, probably primary; eleven, secondary to tumors of the breast, and thirteen secondary to other primary lesions. Pathologically, from 60 to 65 per cent were sarcomas, from 15 to 18 per cent chondromas and 11 per cent carcinomas. Benign tumors other than chondromas have been rare. A few cases included in the series were cases of inflammatory swellings. The difficulties in diagnosis have been the differentiation between benign and malignant tumors. The difficulties in treatment concern the wide opening of the pleura necessary in resection of the thoracic wall, the involvement of intrathoracic structures, such as the lung and diaphragm, and the secure closure of the thoracic wound. The operative mortality has been reduced from about 30 to about 15 per cent; in individual series, it has been as low as 5 per cent. The late results leave much to be desired. The "follow-up" report is given in only sixty-seven of the entire series. In the group of cases of malignant tumors there was freedom from recurrence in nineteen at the time of the last report, but in only three of these did the freedom from recurrence extend beyond a three year period. In the three cases, there had not been a recurrence for four, seven and nine years, respectively. In the group of patients with chondromas, 30 per cent are reported cured, but in many of the cases the period of observation had not extended beyond two years. It is apparent from this summary that the important considerations in this group of tumors are early diagnosis, early radical operation and accurate "follow-up" studies.

MEDIASTINAL TUMORS

Mediastinal tumors include a great variety, which may be divided into benign and malignant tumors.

Benign Tumors or Cysts.—(*a*) Mediastinal Dermoids: After the elimination of duplicates reported by two or more observers and those in which the diagnosis is not established and including two cases of my own not reported, one hundred and thirty-five cases of dermoid cyst or teratoma have been recorded up to January, 1926. Comment on this group of tumors is not necessary, for they are well known. The treatment has varied, and it is of interest to compare the results from the standpoint of the kind of therapy employed. Twelve of these tumors were found at autopsy in persons who had died without the lesion having been diagnosed; eight were diagnosed more or less definitely, but the treatment, if any, and the end-results are not known; forty-six patients died untreated, and in the majority the diagnosis was established by postmortem examination; sixty-nine were subjected to operation. Of the sixty-nine patients subjected to operation, one was treated by simple drainage of the pleural cavity, thirty-four by incision and drainage of the dermoid cyst, twelve by incomplete resection of the tumor and

twenty-two by complete extirpation of the tumor. The results of drainage, partial resection and complete extirpation are as follows: Incision and drainage have been attended by an immediate mortality of 26 per cent and an additional later mortality of 12 per cent, while but 14 per cent of the patients were cured; partial resection has been attended by a total mortality of 16 per cent with cure in 41 per cent, and total extirpation has been followed by a 9 per cent mortality and cure in 91 per cent. It is evident that total extirpation, when possible, is the operation of choice. The results in those cases in which treatment is not given eventually show a high mortality.

(*b*) Other Cysts: Other cysts, such as echinococcus cysts, are rare. Rose's case is one of the few authentic cases. Ciliated epithelial cysts may occur, and about fourteen cases have been reported. These cysts may arise from the thymus, from the trachea or from the bronchi. The majority have been small and without symptoms. The cysts in the cases of Fletcher, of Bramwell and of Sauerbruch (two cases) were large and gave rise to definite symptoms. The only cases in which operation was performed were the two of Sauerbruch. One patient recovered and one died.

(*c*) Benign Connective Tissue Tumors: These tumors include fibroleiomyomas, xanthomas, chondromas or chrondomyxomas, fibromas, lipomas, ganglioneuromas and hour-glass tumors. They have been rare tumors, and have given rise to symptoms of mediastinal compression and a circumscribed shadow in the x-ray film.

(1) Fibroleiomyomas. Only two cases of fibroleiomyoma are reported, both by Jacobaeus and Einar Key. Both tumors occupied the posterior mediastinum; both were successfully removed. I cannot find any tumors with a similar pathologic diagnosis reported elsewhere.

(2) Xanthomas. Again only two cases of xanthoma are reported, one by Wessen and one by me. Both tumors occurred in the posterior mediastinum; both were successfully removed, and the patients have been well for a period of years.

(3) Chondromas or Chondromyxomas. Chondromas or chondromyxomas may arise anteriorly from the sternum and ribs and extend backward into the anterior mediastinum, or from the spine and costovertebral articulations and extend forward into the posterior mediastinum. Occasionally, they may cause a gibbus (Garrè). In two cases, the tumor extended into the spinal canal, giving rise to symptoms of compression of the spinal cord (Nigst). They show a circumscribed shadow in the x-ray film. Up to the present time, only a few patients have been treated surgically. A patient operated on by me died with symptoms of pulmonary embolism after a successful removal. A patient operated on by Garrè recovered.

(4) Fibromas. Eighteen cases of fibroma have been reported. They may involve the pericardium or lie in the anterior or posterior mediastinum. A review of their pathology shows some confusion, and more accurate diagnoses are needed. In the cases of Shen (eleven), Wiedman, Garrè (one), Forni, Ricard and Cokkalis, the tumors were presumably pure fibromas, but the majority of the others suggest invasive qualities as if they might be fibrosarcomas. Operation was performed in only five of the eighteen cases. All the patients recovered after the operative removal of the tumors. The thirteen patients not treated died.

(5) Lipomas. Including Graham's recent case, I have found twelve cases of lipoma in the literature. As Graham perhaps rightly suggests, these tumors may be more frequent than the literature would indicate, because they may, when small, fail to cast a shadow on the x-ray plate. Only three patients have been operated on successfully (Beatson; Sauerbruch; Graham). Von Langenbeck excised the external part of the tumor in Krönlein's case, the child dying of erysipelas. The majority of the patients died untreated. Various authors at the autopsy examination comment on the ease of removal had surgical measures been attempted.

(6) Ganglioneuromas. I have found nine instances of ganglioneuromas in the thorax reported. The tumors arise in the posterior mediastinum in connection with the sympathetic nervous system (ganglions or cords), and pathologically show strands of nerve fibers, often branching, and groups of multipolar ganglion cells. Operation was performed in only three of the nine cases reported in the literature, the remaining patients dying untreated. One of the three patients operated on died and two recovered (Rosensen; Brunner; operations by Lilienthal and Sauerbruch).

(7) Hour-Glass Tumors. The characteristics of the hour-glass tumors are that they arise from the spinal nerves or fibrous structures of the vertebral canal and extend into the mediastinum, or arising in the thorax (sympathetic chain, spine, ribs, etc.), they extend into the vertebral canal. Because of their growth through an intervertebral foramen, they have an hour-glass shape. They may produce symptoms of cord compressions, of mediastinal compression, or of both cord and mediastinal compression. I have found eleven cases in the literature and have two cases of my own. The condition is probably more common than the literature would indicate, for Cushing, Elsberg and other neurosurgeons have told me that they have observed cases but have failed to report them. Pathologically, the tumors have been diagnosed as neurinomas, neurofibromas, fibromas, fibrosarcomas and chondromas. Operation was performed in eleven of the thirteen cases. In eight, the

primary approach was directed to the spinal lesion; in three, to the thoracic lesion. In all eight cases in which a primary laminectomy was done, the intraspinal tumor was removed, and, with two exceptions, the mediastinal lesion also. In the three cases in which the primary approach was mediastinal, the mediastinal tumor was removed, but not the intraspinal. There were six recoveries and four deaths, and in one case the result is unknown. Of the six patients who recovered, one was well nine years; one, eight years, and one, four months after operation. Death was due to meningitis in one case and apparently to shock in three.

As one reviews the literature on this group of benign tumors, it is obvious that the surgical treatment has been satisfactory. The great difficulty lies in the differential diagnosis. Rarely has a positive diagnosis of the nature of the lesion been made, but it has been possible to make a diagnosis of benign tumor in many cases.

Malignant Tumors.—(*a*) Lymphoma, Lymphocytoma, Lymphosarcoma, Hodgkin's Disease: There is a great deal of confusion in the literature regarding the various conditions arising from lymphoid tissues, and it becomes evident on going over the case reports of mediastinal tumors that no clear idea may be had of the frequency, origin and histologic picture of the various lesions arising from them. Only comparatively recently has the attempt been made to trace the origin of these tumors to the various elements of lymphoid tissue and to the thymus gland.

(1) Lymphoma. The simple lymphoma described by Ewing, occurring in the neck, axilla and groin, is rare in the mediastinum, and I have failed to find an undoubted case in the literature.

(2) Lymphocytoma. Under the general term of lymphocytoma, a number of cases of mediastinal new growth have been described in the literature. For the most part they have been called malignant lymphadenoma or malignant lymphocytoma. Tumors associated with lymphatic leukemia or pseudoleukemia have occurred in the mediastinum but they are rare. In general, they have been grouped under the term leukosarcoma. In a series of cases collected by Fabian, Friedlander and Foot, a malignant tumor of the thymus has been associated with a leukemic blood picture, so that leukemia with involvement of the thymus has come to be recognized as an atypical and malignant variety of tumor.

The lymphocytomas as compared with lymphosarcomas and Hodgkin's granuloma are rare tumors. The pathologic basis for the group is the predominance in the tumors of small cells resembling lymphocytes, but whether these small cells are derived from lymph nodes or thymus is not often clearly stated. As a rule, the tumors arise in the anterior

mediastinum, often in the general location of the thymus. They vary in size and may come to fill the space between the sternum and spine and surround the trachea, great vessels and esophagus. They invade the pleura and lungs and metastasize widely.

(3) Lymphosarcoma. Lymphosarcoma must be considered as one of the commonest of mediastinal tumors. In a series of sixty cases of malignant mediastinal tumor which came to autopsy, Ross found forty-four sarcomas and ten carcinomas. Thirty-two of the forty-four sarcomas were lymphosarcomas. In the literature the reports of cases number in the hundreds, but in many the diagnosis is clinical and histologic proof is lacking. The origin of these tumors is not always clear. They may arise either from the mediastinal lymph nodes or from the thymus gland. In the earlier literature it is impossible to trace the origin of the tumors, in the majority of cases, but with the interest aroused in the pathology of the thymus gland, the distinction between tumors of lymph node and thymus is being more commonly made. Ewing is of the opinion that perhaps the majority of the mediastinal lymphosarcomas have a thymic origin.

Like the lymphocytomas, the lymphosarcomas grow rapidly, fill the mediastinum, surround the great vessels, trachea and esophagus, extend to the pleura and lungs and metastasize to various organs. They are rapidly fatal, the average duration of the disease in Ross' series being eight months.

The surgical treatment of the lymphosarcomas and lymphocytomas has been unsatisfactory; but it is to be said that surgical measures have been employed in only a few cases. The disease progresses so rapidly that surgical intervention has been impossible at the time patients have come under observation. X-ray and radium therapy has been the treatment of choice up to the present time. It is well known that these tumors respond favorably to radiotherapy, and a few reports of freedom from recurrence for as long as four years are at hand.

(4) Hodgkin's Disease. Rarely does Hodgkin's disease begin primarily in the mediastinum. The condition is rather well understood. The clinical manifestations are fairly definite; the x-ray shadow is usually suggestive. The treatment used at the present time is radiotherapy.

(*b*) Thymic Tumors: I shall omit from consideration at the present time the rare tumors of the thymus, such as cysts, myxomas and lymphangiomas; the thymic hyperplasias that occur in persistent thymus and status lymphaticus; the thymic enlargements occurring in association with exophthalmic goiter, and also the thymic enlargements and tumors seen in association with myasthenia gravis. I shall confine my remarks only to the primary malignant tumors of the thymus.

There is a great deal of uncertainty regarding the origin of certain cell elements of the thymus, which has resulted in difficulties in the satisfactory classification of thymic tumors; as one reads the literature, one is confused by the variety of names given them. Most authors agree, however, that two main groups of malignant tumors may arise in the thymus, sarcoma and carcinoma. Ewing divided the thymic tumors into lymphosarcoma or thymoma, carcinoma and sarcoma, and it is the classification that I have followed. Some uncertainty also exists as to which tumors arising in the mediastinum may fairly be called thymic tumors, and various authors have stated certain characteristics which they must fulfil.

A study of the literature shows less than 100 cases of malignant thymic tumor reported. In 1911, Rubaschow compiled sixty-nine cases from the literature, and since then various authors have reported from one to four cases. Foot, in his last report (January, 1926), stated that if the miscellaneous cases of Rubaschow are excluded, there are eighty-one cases of primary malignant thymic tumor reported. The majority of these are lymphosarcoma or thymoma, sarcoma and carcinoma occurring more rarely. In general, they resemble the lymphosarcomas previously noted, both in the rapidity of their growth and in their clinical manifestations. In no case has an attempt been made to remove the mediastinal portion of the tumor, although external tumors the result of extension from the primary growth have been removed. X-rays and radium have been used in a small number of cases. In Janeway's series, no improvement followed in three of the four verified cases; in one, there was temporary improvement.

(*c*) Sarcoma and Carcinoma: The sarcomas and carcinomas other than those mentioned form a miscellaneous group and in many respects are also shrouded in obscurity. The sarcomas may arise from the connective tissue elements of the mediastinal lymph glands and of the thymus and from the connective tissues, tendons and periosteum of the sternum, ribs and spine. The carcinomas other than those arising from the reticulum cells of the thymus are the result of extension from primary carcinoma of the lung, trachea, bronchi, breast or esophagus, or are metastatic growths from primary carcinomas elsewhere. It is certain that primary carcinoma of the mediastinum is a much rarer condition than the earlier literature would indicate. Between 1896 and 1901, Lorisch was able to find only two cases of carcinoma reported; between 1901 and 1907, Christian was able to find but one satisfactory case. As one reads the literature on this subject, it becomes apparent that an attempt to assemble the cases at the present time would be unprofitable, because of the lack of definite information regarding the origin and pathology of these tumors.

When studies of the malignant tumors of the mediastinum are reviewed, one must conclude that there is great opportunity for advancement in the knowledge concerning the origin, pathology, early diagnosis and treatment of these tumors. It is one of the group of thoracic tumors which requires much further elucidation.

Tumors of the Pleura.—I shall omit discussion of the echinococcus and other cysts and of the rare benign tumors, such as lipomas, fibromas, enchondromas, chondromyxomas, etc., and speak of the primary malignant tumors. In the literature, these tumors are grouped as endothelioma, carcinoma and sarcoma, but when one attempts to differentiate them one finds this almost impossible. The synonyms for endothelioma (carcinoma, sarcocarcinoma, endothelial carcinoma, pleuritis carcinosa, lymphangitis carcinomatodes, mesothelioma, etc.) perhaps show as well as anything the confusion which exists among pathologists concerning this type of tumor. Before 1870, a number of authors recognized tumors of the pleura which they called cancers of the lung; others of the same period opposed the idea of primary cancer of the pleura and considered them secondary to tumors elsewhere. In 1870, Wagner suggested that the tumor cells in his case arose from the endothelial cells of the pleural lymph vessels and called his tumor an endothelial cancer. This idea governed the major number of theories until 1890. In 1891, Engelbach suggested that the tumor cells in his case arose from the surface cells of the pleura rather than from the endothelium of the lymph vessels; from this time on pathologists have considered the possibility of tumors arising from both sources. Benda, in agreement with Hertwig's conception that the pleural cavity is derived from the primitive celom and is a true epithelium, concluded that his tumor must be called a primary carcinoma. The possibility of two tumors, one a carcinoma, one an endothelioma, arising primarily from the pleura is therefore suggested. Ribbert, after a discussion of various views, concluded that all or practically all primary tumors are carcinomas and would eliminate the term endothelioma. Demole, after a careful analysis of the reported cases of endothelioma of the pleura, concluded that most of them represent carcinomas, either primary or secondary. Subsequent writers have called the tumors in their cases carcinoma. DuBray and Rosson, following Adami, proposed the term primary mesothelioma for the tumor in their case, basing their decision on the opinion that the primitive celom and therefore the serosal cells are derived from the mesenchyme. They do not see the justification for considering these tumors carcinomas, and the term endothelioma to them is not appropriate. Some subsequent writers, following this lead, have designated the tumors in their cases as mesotheliomas. Some later writers, especially Robertson, denied that primary endothelioma or carcinoma or mesothelioma of the pleura occur at all, but that the tumors represent

metastases or extensions from a primary carcinoma of the lung or other organ. According to Robertson, only the sarcomas can be classified as primary malignant tumors of the pleura.

It must be evident that this whole subject requires further study, and chiefly, by the most careful examinations at autopsy. It is possible that pathologists have been dealing with a composite group of tumors all of which may and have given rise to an identical clinical picture. To state dogmatically that there is no such thing as endothelioma and that all these tumors represent extensions or metastases from carcinoma, for example, would be unwise at the present time, for there is not sufficient evidence. In a recent case of my own which presented the histologic picture of endothelioma, carcinoma of the lung was not found, nor did the most painstaking examination reveal carcinoma anywhere in the body.

Whatever term is applied to these tumors, there are considerably over 100 cases reported in the literature. The tumors occur in two forms; they are diffuse and circumscribed. The former first appears in the form of multiple nodules or flat elevations which fuse; the pleura becomes thickened and eventually is converted into a diffuse, firm, opaque mass covering and compressing the lung. The condition is associated with a bloody exudate. The circumscribed form gives rise to globular tumor masses of varying size which cast a circumscribed shadow in the x-ray film. I have seen three instances of the diffuse variety and four of the circumscribed. It should be said with reference to the possible origin of the latter tumors that in no instance was there any physical or x-ray evidence of carcinoma of the lung. The diffuse variety does not lend itself to any form of treatment so far as I am aware, and is invariably fatal. In the circumscribed form, surgical intervention offers some hope of relief. The tumor was operable and was successfully removed in three of the four cases which I have seen. Recurrence took place in one in ten months; there was freedom from recurrence in one for over two years, and in one the time elapsed has been less than one year. I find few records of operations for this condition other than my own.

From what has been said of the endothelioma, it would be rather useless to discuss the sarcoma of the pleura. In any compilation of reports of a large series of pleural tumors, sarcomas are included. Block, for example, collected sixty-three cases of presumably authentic pleural tumors, of which forty-seven were considered endotheliomas and sixteen sarcomas. Some authors maintain that the sarcoma is pre-eminently the primary malignant tumor of the pleura; others have eliminated it and have grouped it under endothelioma.

The secondary or metastatic sarcomas and carcinomas of the pleura require no comment.

Cysts and Tumors of the Lungs.—(*a*) Echinococcus Cysts: There is a voluminous literature on the subject of echinococcus cysts, and a number of authors have personally observed over 100 cases. The condition continues to be rare in the United States, most of the reported instances being in foreigners recently arrived in this country. Those familiar with the disease group cases symptomatically into (*a*) those with silent or latent cysts, (*b*) those with few or no symptoms but with definite physical signs, (*c*) those with definite symptoms and physical signs and (*d*) those with cysts complicated by infection. The diagnosis is unhesitatingly made in those countries in which the disease is common on the symptoms, signs and x-ray picture; but when uncommon it may obviously be difficult to differentiate echinococcus disease from dermoid cyst or benign tumor before rupture into a bronchus has occurred. In the diagnosis or treatment, the majority of authors still affirm the dangers of aspiration. According to some authors, spontaneous evacuation through a bronchus results in a cure in from 80 to 100 per cent of the cases (Lepicard, Billard, Morquio); others have found different results, stating that two thirds of the patients not treated surgically eventually die of the disease. The surgical treatment consists of pneumotomy, ideally with total enucleation of the intact cyst; otherwise, evacuation and drainage are employed. Guimbellot compiled 223 cases in which operation was performed with 194 recoveries and 29 deaths; Garré compiled 99 cases in which open operation was performed with cure in 80 per cent. Tuffier reported thirty-five cases with one death. The end-results are good, although an occasional recurrence has been reported.

(*b*) Other Cysts: Other cysts such as dermoids, congenital cysts, etc., may be passed over. The former have been discussed under mediastinal tumors, the latter have little surgical interest.

(*c*) Benign Tumors: Benign tumors such as fibromas, lipomas, enchondromas, osteomas, angiomas, lymphomas and adenomas have all been described. They are rare and few have been subjected to surgical measures. Borelius and Sjovall found accidentally by routine x-ray examination a round, sharply defined shadow the size of an apple in the lower lobe of the left lung. At operation, a hard spherical tumor which lay definitely within the lung tissue was enucleated. Microscopically, it was a mixed tumor containing cartilage, fat, muscle bundles and epithelium.

(*d*) Malignant Tumors: (1) Endothelioma. I shall pass over the endothelioma. The same discussion regarding them as has been noted under pleural endothelioma occurs in the literature.

(2) Primary Carcinoma. Up to the present time there have been more than 700 cases of primary carcinoma reported. In recent years,

the majority of writers on the subject have found an increase in the frequency of pulmonary carcinoma. It was predicted by some that following the influenza epidemic during the war there would be an increase in the incidence of pulmonary cancer, and this actually has been true. But it has been pointed out that no similar increase in pulmonary cancer followed the pandemics of 1890-1894; that in the majority of the present cases of carcinoma a previous history of influenza is lacking; that the rise in frequency began as early as 1900, and that the past epidemic of influenza affected males and females about equally while pulmonary carcinoma has occurred four times as frequently in males.

A second group of causative factors which have been suggested as possibly responsible for the increase are physical agents, viz.: the more frequently use of x-ray for diagnosis, the increased production of dust and smoke in cities, etc. Still a third group of possible agents are chemical, particularly the coal tar derivatives, which are so much more prevalent since the advent of tarred roads and the automobile. Whatever the cause of the increase, according to some, the peak has been passed and will be followed by a decline in the number.

Pathologically, there are three types of pulmonary carcinoma, those arising from the bronchial epithelium, from the mucous glands of the bronchi and from the alveolar epithelium. The first two make up the large proportion of tumors.

Great advances have been made in the recognition of the symptoms and physical signs of pulmonary cancer, and whereas previously the diagnosis was not often made, it is now made often and fairly easily. The x-ray manifestations are also more often recognized; bronchoscopy has aided not a little in cases in which the tumor projects into a bronchus.

The treatment is still unsatisfactory. X-rays and radium have been repeatedly used, with some reported instances of improvement or checking of growth and an occasional from three to five year symptomatic cure with disappearance of the shadow in the x-ray film. About thirty patients with primary malignant tumor of the lung (including sarcomas) have been subjected to surgical procedures. In many of these cases, operation consisted merely in the drainage of associated bronchiectatic cavities or empyemas, or in the removal of tissue for diagnosis. In seventeen instances, however, an attempt was made to remove the tumor completely, and although eleven of the patients died soon after operation, several encouraging results have been obtained. One of Lenhart's patients lived one and one-half years; one patient operated on by Reid lived two years and two of Sauerbruch's patients lived three and five years, respectively. The possibilities of cautery pneumectomy as advocated by Graham in chronic pulmonary suppuration should be remembered in this connection.

(3) Primary Sarcoma. From a review of the literature it would, I think, be unprofitable to discuss primary sarcoma of the lung. It is another subject which requires much elucidation.

(4) Metastatic Carcinoma and Sarcoma. These growths as is well known, form by far the most common malignant tumors of the lung. In 10,829 autopsies at the pathologic institute of Munich, Seydel found 1,342 tumors of which 184 involved the lungs and pleura; 74 per cent were metastatic, 9 per cent extensions from tumors in adjacent areas and 16 per cent primary. It will be unnecessary to comment on them. They are readily recognized by the roentgenograms. Some respond well for a time to the x-ray and radium therapy; others are not influenced. One is lost as to how to proceed when they do not respond.

SUMMARY

Such, then, is a summary, of necessity incomplete, of the surgical aspects of thoracic tumors. It is evident, I think, first, because of the confusion which exists regarding the origin, pathologic characteristics and terminology of many groups of intrathoracic tumors, and second, because no one man sees a sufficient number of cases, that for a time there should be a thoracic tumor registry. It must also be evident that further aids are needed in the differential diagnosis of intrathoracic tumors. I have indicated that surgical treatment in cases of benign tumors has been eminently satisfactory; it could be made much more satisfactory if benign lesions were treated earlier. The literature is full of reports of lost opportunities; of tumors found at autopsy which could easily have been removed. A change of sentiment among physicians and patients regarding the advisability of thoracic exploration is necessary. It can be shown that thoracic exploration at present is a procedure attended by a low mortality; and yet within the past few years, Dr. Reid and I, for example, have seen eight cases of benign tumors in which the patients refused operation either because of the absence of serious symptoms or because of their own or their physicians' fear of operation. Finally, accurate follow-up studies need to be made if the results of surgery are to be known.

THORACIC TUMORS

A ROENTGEN STUDY *

J. J. SINGER, M.D.
ST. LOUIS

During the past few years, there have been many important advances in the diagnostic method of thoracic conditions. Among the more valuable additions may be mentioned the use of iodized oil and of diagnostic pneumothorax. Scarcely less important is the selection of the position most favorable for obtaining information in fluoroscopic and roentgen examination. Any one or all of the new methods may be applicable in the individual patient. It has been possible for us to combine these methods in a most satisfactory manner because of the existence of the chest service of Barnes Hospital where there is a specially trained personnel and all necessary equipment for the most detailed examination.

In no class of cases is the proper use and selection of methods more necessary than in the diagnosis of tumors of the lung.

This study is concerned with a series of tumors observed by Dr. Evarts Graham and myself in the chest service. It is my chief aim to illustrate how various advanced methods of diagnosis may be combined to demonstrate the presence of tumors. We have often been forced to content ourselves with a recognition of the presence of tumors without being able to state to what type they belonged. The differentiation of tumors even with present methods offers the greatest difficulty.

Barjon,[1] in his book, attempted to define the roentgenologic characteristics of different types, and included a detailed description of primary cancer, including the lobar and hilum forms and of secondary cancer, both nodular and diffuse. Carman,[2] on the other hand, was willing to state that the only positive evidence of carcinoma in his experience was an extensive dense lobar shadow attended by smaller shadows of metastasis.

For the purpose of this radiographic study, we have classed as tumors all growths, whether malignant or benign. Even old healed abscesses have occasionally been classified because, radiologically, we can not with certainty distinguish them.

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* From the Department of Internal Medicine, Washington University School of Medicine and Barnes Hospital, Chest Service.

1. Barjon: Radio Diagnosis of Pleuro, Pulmonary Affections, New Haven, Conn., Yale University Press, 1918, p. 143.

2. Carman: Carcinoma and Sarcoma of the Lung, M. Clin. N. Amer. **5**:352 (Sept.) 1921.

DIAGNOSTIC PNEUMOTHORAX

By the use of diagnostic pneumothorax and thoracentesis, one can often classify shadows so that the true nature of the condition may be recognized. In cases which show evidence of complete density in the whole or in part of the lung fields and when physical signs indicate fluid, a diagnostic thoracentesis is done. If fluid is obtained, a part of it is replaced with air. Occasionally, diagnostic pneumothorax is done even

Fig. 1 (case 1).—Erect position (after withdrawal of small amount of fluid and replacing with air). An air pocket is seen outlining the lower portion of the lung which is completely dense and collapsed.

when there is no evidence of fluid in order to determine whether tumor density exists in the lung or in the pleura itself.

The first case exemplifies the practical use of this method.

CASE 1.—C. S., aged 65, a woman, white, entered Barnes Hospital on Feb. 23, 1924. For several months she had pain in the right side of the chest with a dry hacking cough. The temperature was from 99.5 to 100 F. There had been a moderate loss of weight. Aspiration had been performed many times by her former physicians, and the fluid was always serous. For several weeks before admission to our service, aspirations had been repeatedly attempted, but no fluid had been obtained.

Physical examination revealed over the right side of the chest below the second rib, flatness to percussion, absence of breath sounds and no râles. In the left lung, there was compensatory breathing. Fluoroscopic and roentgen examinations of the chest showed a dense shadow throughout the right side below the second rib. Lung tissue could not be seen through the density. The right side of the diaphragm was also lost in the shadow.

This history, age of the patient and the absence of tubercle bacilli in the sputum indicated a malignant tumor, but the roentgenogram and the dry taps were confusing.

A diagnostic thoracentesis showed clear fluid; 200 cc. was removed and partly replaced with air (diagnostic pneumothorax). The patient was then examined fluoroscopically.

The air pocket now showed the various structures in the right side of the chest clearly. A definite shadow was noted in the third and fourth interspaces, and the lung below was found to be adherent in such a position that fluid was inevitable. A diagnosis of tumor of the lung was made. An exploratory operation was done by Dr. Graham. Tissue was removed which on section revealed a carcinoma arising from bronchial mucous membrane and metastasizing to the pleura.

By employing artificial pneumothorax, we were able in this case to change an obvious radiologic diagnosis of fluid to that of a tumor of the lung, a premise later proved by operation.

POSITIONING

By positioning, we mean putting the patient in various positions before the fluoroscope to bring out the most suitable posture to show the condition of the lungs.

Case 2.—C. A. P., a white man, married, entered Barnes Hospital on Sept. 7, 1925. For one month he had complained of sharp pains about the right costal margin. This was accompanied by a cough productive of a purulent expectoration which occasionally had a foul odor. He had lost 10 pounds (4.5 Kg.). One sister and one brother had had tuberculosis.

At the base of the right lung there were diminished breath sounds but no impairment of resonance. A few low pitched râles were heard over this area. The whispered sounds were slightly increased in the right interscapular area. The roentgenogram showed nothing definite, although the bronchial branchings were somewhat thickened.

In the fluoroscopic examination, by placing the patient in various positions, we found a dense mass hidden in the diaphragmatic sulcus. When the position most favorable for demonstrating the mass had been obtained, a roentgenogram was taken.

With this information, a diagnosis was made then of tumor or of abscess of the lung with fibrosis. The latter suggestion was made because in another similar case a mass, removed at operation, proved to be a fibrosed abscess.

It will be noted in this case that the dense shadow in the right diaphragmatic sulcus would have been lost in the position ordinarily used in routine roentgenologic examinations. Positioning enabled us to recognize the mass. The history suggested an abscess.

IODIZED OIL 40 PER CENT IN DIAGNOSIS

Iodized oil 40 per cent instilled into the lung by one of the many methods described in a former paper,[3] but especially by the simple aspiration method, often outlines a tumor mass, shows stricture of the bronchi and helps to differentiate the tumor mass from the lung itself.

It might be added here, in hundreds of examinations with iodized oil in the lungs, we have never seen any bad effects on the patient. There

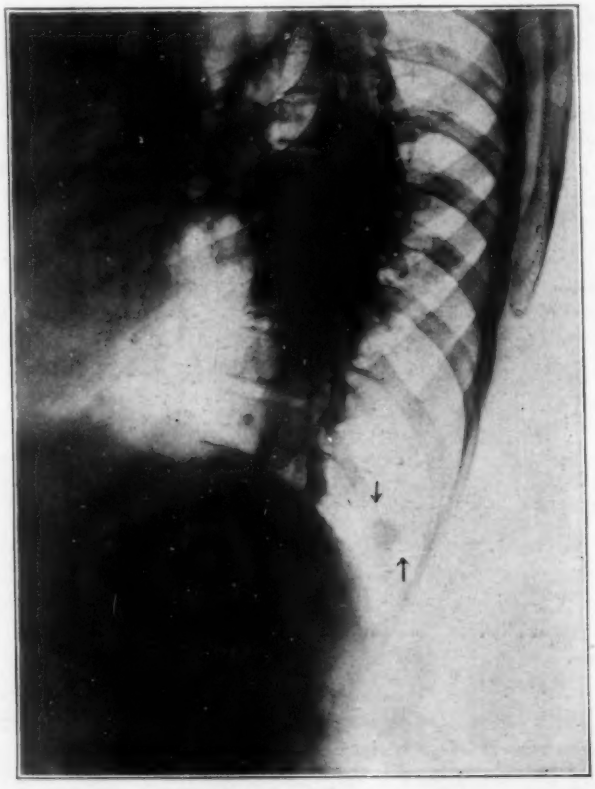

Fig. 2 (case 2).—Value of position. A small dense shadow is seen in the right costophrenic angle. In the original roentgenogram, this shadow was lost in diaphragmatic density.

have been reports made by other workers in this field who have obtained some reactions.[4] Many of the cases reported were in patients with acute tuberculosis in whom the iodized oil was introduced by means of a bronchoscope; we never use iodized oil in this form of disease and certainly do not recommend its introduction with a bronchoscope.

3. Singer, J. J.: Simple Method of Introducing Iodized Oil in the Lungs, J.A.M.A. **67**:1298 (Oct. 16) 1926.

4. Archibald and Brown: Dangers of Introducing Iodized Oil into the Tracheo-Bronchial System, J. A. M. A. **88**:1310 (April 23) 1927.

CASE 3.—J. E. W., aged 34, white, a minister, entered Barnes Hospital on April 17, 1924. He complained of pain in the right side of the chest and dyspnea. According to his family history his father had died of cancer of the stomach; the grandfather and greatgrandfather had also died of cancer. The onset of the present illness was in October, 1923, when he had a pain in his right shoulder and in the right side of the chest in the region of the third and fourth ribs. This disappeared in a few days only to return in two weeks. He had seven such attacks. In March, 1924, he had a severe attack of pain, dyspnea and unproductive cough. He had some difficulty in swallowing in November, 1923, but this disappeared. The pain extended into the right arm.

Fig. 3 (case 3).—Value of iodized oil. A large mediastinal shadow is seen through which the normal bronchial tree was outlined (the negative showed this much clearer).

Physical examination showed numerous firm, slightly painful subcutaneous nodules scattered over the entire body. There were signs of fluid in the right side of the chest, with pulsation on anterior wall of the chest over the second interspace. Seven hundred cubic centimeters of clear yellow fluid was removed and replaced by 400 cc. of air. A pulsating mass was then recognized in the mediastinum projecting into the right side of the chest and indicating aneurysm of the first portion of the aorta.

Examination of the blood showed a four plus Wassermann reaction. The diagnosis made from roentgen examination was pleurisy on the right side with effusion; pleural tumor; aneurysm of the first portion of the aorta; bronchial

block. In the light of these observations, the clinical diagnosis was aneurysm of the aorta, the first portion; chronic bronchitis, syphilis and hydrothorax.

The patient was placed on mild antisyphilitic treatment (potassium iodide and mercury) and showed improvement. When the patient was admitted to the hospital at another time, iodized oil was injected into the right lung by the aspiration method. The roentgenogram showed a large mediastinal tumor through which was seen a perfect bronchial tree; this showed radioscopically that the lung itself was not involved and had not been compressed. The character of this tumor was not definitely diagnosed although it was suggested that it might be a fibroma; the

Fig. 4 (case 4).—Value of biopsy. A large mediastinal shadow is seen; a gland removed from the right cervical region showed lymphosarcoma. After one massive roentgen treatment the mass entirely disappeared.

patient showed numerous fibromas on the skin (von Recklinghausen disease). As he also had a four plus Wassermann reaction, aneurysm must be considered (no pulsations were ever noted fluoroscopically).

BIOPSY AND OTHER METHODS AIDING DIAGNOSIS

In the next two cases, I will attempt to show the value of biopsy and other laboratory examinations, especially basal metabolism.

CASE 4.—O. J. S., aged 34, a man, white, a stove molder, entered Barnes Hospital on Nov. 9, 1925. He complained of swelling of his neck, face and wrist,

of pain in his shoulder and of cough with dyspnea. Three weeks previous to admission the glands in the neck begun to enlarge rapidly. Swelling of the face, eyes and the left wrist appeared later. Pain in the shoulder followed and he then became short of breath, and developed an unproductive cough. He did not feel sick and there was no fever.

Physical examination revealed a fairly well developed man, slightly dyspneic. Painless, lymph nodes, firm, enlarged and movable, were found in the anterior cervical region, in the axillae, at the angle of mandible and in the inguinal region. There was an increased substernal dulness at the level of the second and third

Fig. 5 (case 5).—A dense shadow is seen in the upper right side of the thorax. A definite diagnosis was made only after operation for thyroidectomy and removal of section of the mass below the clavicle; hyperthyroidism, substernal thyroid.

intercostal spaces. The breath sounds were diminished on the left side, anteriorly and posteriorly, and there were many loud moist râles. The right side was not abnormal.

Fluoroscopy and roentgen examination revealed a large mass completely filling the upper chest cavity, when seen in the anteroposterior position. There was no pulsation. The apex of the heart was distinctly seen only when the patient was turned tangentially. The laboratory observations were all negative. It was reported that biopsy performed on one of the cervical glands showed lymphosarcoma.

The patient had one roentgen treatment. A later roentgenogram of the chest showed a marked diminution in size of the shadow and improvement in the general condition.

In this case a dense shadow was seen, probably that of a tumor, the nature of which is indefinite. Biopsy of the cervical gland showed lymphosarcoma.

Roentgen treatment in massive dose caused a rapid disappearance of the mass in a few days.

CASE 5.—S. F., aged 48, a woman, white, married, entered Barnes Hospital in December, 1925. The chief complaints were those of weakness, and loss of weight. Her general health had been good until five years before. She had sinus trouble for the past five years. The onset of her present condition was an attack of influenza five years previously when she had been in bed for six weeks. A few

Fig. 6.—Value of iodized oil. A tumor mass is seen in the upper lobe of the right lung with obstruction of the bronchus, outlined by iodized oil. (The patient was tipped with head downward for the picture.)

months later, the veins in her chest became prominent. She had failed steadily, had had dyspnea and had lost weight for the past year. She was given massive roentgen treatments, but her condition became worse and she became weaker; she lost 22 pounds (10 Kg.). She also coughed up phlegm in the mornings.

Physical examination showed tenderness over the sinuses. There were enlarged veins over the right side of the neck and part way down the arms. There was dulness in the first, second and third intercostal spaces, extending from about 5 to 6 cm. to the right of the sternum. The whispered and breath sounds over this area were increased.

Laboratory examinations showed: normal urine; red blood cells, 4,400,000; white blood cells, 5,200; hemoglobin, 75 per cent; differential count, normal. The Wassermann and Kahn tests were negative. The blood pressure was 125 systolic and 70 diastolic.

The mass in the upper right side of the chest extended almost to the anterior axillary line; there was no bruit or pulsation nor marked changes in the physical signs. The mass seemed larger than before treatment. Hodgkin's disease, thymus and lymphatic mass were also ruled out of the diagnosis because of the negative results of massive roentgen-ray therapy.

Fluoroscopic and roentgen-ray examination showed a dense mass in the upper right side of the chest extending to the anterior axillary line; pulsations were not noted.

The probable diagnosis was substernal thyroid, mediastinal dermoid and malignancy.

In this case a large tumor was seen in the upper mediastinum; the shadow seen could indicate aneurysm, lymphosarcoma, Hodgkin's disease, cyst or benign tumor. The laboratory examination, metabolism test, + 25, and history showed evidence of toxic thyroid.

At operation, the thyroid was removed, and a section of the mass below the sternum proved it to be thyroid tissue.

In this case one was able to make a diagnosis of substernal thyroid, because of the aid given by the laboratory tests.

CONCLUSIONS

1. Thoracic tumors radiologically show rather dense shadows, the nature of which must be determined by a complete study of physical signs and history.

2. The newer aids in diagnosis, such as position, biopsy and iodized oil, bronchoscopy, thoracoscopy, diagnostic pneumothorax and laboratory tests are invaluable in bringing out the true character of the shadows.

3. In patients past 40 years of age who show signs of abscess of the lung, one should suspect a malignant condition if the condition has been present for several months. Sections of the tissue should be removed to substantiate the diagnosis.

Beaumont Building

CYST OF THE LUNG

RECOVERY FOLLOWING OPERATION FOR PERMANENT DRAINAGE

HOWARD LILIENTHAL, M.D.
NEW YORK

Mrs. Sophie S., aged 54, came to me on Nov. 5, 1927. Other than typhoid during adolescence, her past history was irrelevant. The bowel movements were regular up to two months before I saw her, when she became constipated. For nine years, there was pain in the chest on exertion, but there never was any cough or expectoration. About six months before her visit, she began to have "nervous spells" at night with pain in the chest so that she awoke in fright. She went to the Jefferson Hospital in Philadelphia, where aspiration of the upper left part of the chest was performed, and several ounces of chocolate-colored fluid were removed. An attempt was made to relieve a resulting pneumothorax, but with little success. The nocturnal attacks continued. These spells—the patient objected to the use of the word attacks as not clearly describing the condition—occasionally came on during the day as well as at night. There was complaint of difficulty in swallowing, especially of solid food.

Examination of an x-ray film which the patient brought revealed a large globular mass in the upper left part of the chest extending to the level of the ninth rib posteriorly. The heart was pushed more than two fingerbreadths into the right side of the chest.

A report from Jefferson Hospital Clinic, dated Nov. 10, 1927, stated that the material which was withdrawn by aspiration, on microscopic examination, was found to contain "many red cells, disintegrated leukocytes and a large number of pale cells with prominent nuclei." The description of the roentgen-ray appearance stated that the mass showed a rounded border below and on the mediastinal side, and that it displaced the heart, trachea and aorta toward the right. Bronchoscopy revealed a bulging of the left wall of the trachea which produced moderate stenosis a short distance above the carina. Abnormal pulsation was not seen; ulceration was not noted nor was any resistance shown to the passage of the bronchoscope. The bulging area seemed soft and capable of displacement to the left. "The bronchi and their subdivisions appear normal." The report of the examination of fluid from this cyst by Dr. Louis Gross, director of the laboratories at Mount Sinai Hospital, stated that there were "many peculiar globular masses of bluish staining fluid containing red cells." Further x-ray studies were made at Mount Sinai Hospital, but there was no change in the size of the mass. It was thought to be adherent to the anterolateral wall of the chest at the level of the second interspace, and thence upward in the anterior axillary line. This, by the way, proved to be an error. The mass was in contact with the wall of the chest in this area, but was not adherent.

The patient's general condition was fair. There was no fever and she was up and about. The systolic blood pressure was 120. Examination with a thick medium within the esophagus showed this organ pushed to the right and constricted (figs. 1 and 2).

A diagnosis in this case was extremely difficult, but I strongly suspected dermoid cyst in spite of the absence of hair or other dermoid material from the aspirated fluid. Dermoid cyst or teratoma had also been diagnosed at Jefferson

Hospital. It should be noted, however, that the mediastinal side of this globular mass has a greater convexity than is usually seen in tumors of this type arising in the mediastinum.

I concluded that an attempt to marsupialize the cyst should be made, and that later a more radical procedure might be undertaken.

Mrs. S. entered Mount Sinai Hospital on December 18, where the usual clinical tests and physical examination revealed nothing abnormal except the thoracic mass. Three days later, using local anesthesia supplemented by a little nitrous oxide, I operated on her.

A longitudinal incision was made in the left anterior axillary line dividing some of the fibers of the pectoralis major and exposing the second rib. About 1½ inches (3.7 cm.) of this rib was resected, and it became evident from the freedom of motion of the pleura that adhesions would not be found here. Nevertheless, I opened the chest by an incision about 2 inches (5 cm.) long and at once

Fig. 1.—Congenital cyst of the left lung encroaching on the mediastinum with obstruction of the esophagus. Note the almost globular tumor with equal convexity of mediastinal and parietal contours. Dec. 16, 1927. (Stereoscopic technic.)

saw the cyst, its thin walls bluish in color and traversed by large superficial vessels. The location of the mass was apparently in the substance of the upper lobe of the lung. It was very soft on palpation. Puncture with a fine needle yielded brownish fluid which on examination revealed for the most part broken-down red cells, a few white cells and other cells which looked like large undifferentiated epithelium. This was from the report of a clinical examination not made in the laboratory. Only a few drops of fluid could be aspirated through the fine needle that was used. During the latter part of this operation it had been necessary to administer a little nitrous oxide, the patient having shown signs of distress. The pleura was then walled off with a packing of gauze surrounding the presenting surface of the cyst and another piece of gauze was laid on this surface and marked with a safety pin. The chest was then closed over the packing, without drainage, using a fine silk suture. Scarcely any blood had been lost, and shock was not expected. Less than twenty-four hours later, however.

the respirations had become extremely rapid, and the patient complained of severe pain in her back. The pulse rate had risen to about 120. She was at once taken to the x-ray room, where fluoroscopy revealed great dislocation of the mediastinum and much fluid in the chest. About thirty hours after operation, I placed a small flapper-valved drainage tube into the lower part of the chest through a cannula.

Fig. 2.—Same case as figure 1, but in oblique position; deformed esophagus shown by thick barium meal. Dec. 16, 1927.

Fig. 3.—The cyst has been drained by transpleural operation. The shorter tube lies within the cyst, the outer one is outside the cyst and curves on itself within the pleural cavity. Drainage is not complete and a fluid line is seen below. The picture very much resembles that of a hydropneumothorax. Jan. 13, 1928.

Air and a few ounces of bloody fluid escaped, but the patient was relieved, only to become dyspneic again twenty hours later. Drainage occurred slowly through the tube; the temperature had risen to 102.5 F., and the respirations were 40 a minute.

Dyspnea was progressive with still further pushing of the mediastinum to the right and increased difficulty in swallowing. The following day, I opened the upper wound and inserted a large tube into the pleural cavity. This tube was made airtight with adhesive plaster, and a finger-cot valve was applied. There was immediate relief, respirations becoming easier, and the temperature dropped. The wound, naturally, suppurated, and x-ray examination showed the cyst more than half filled with fluid. I was, therefore, obliged to operate again in order to secure dependent drainage of the cyst, and for this general anesthesia was employed. A long eighth interspace incision was made, and with the aid of the ribspreader the cyst was easily seen and brought into perfect view by stripping away the adherent lung. There was no general empyema, the suppuration having been confined to the upper part of the chest. In the lower lobe, which otherwise looked normal, near the costophrenic sinus, there was a globular mass about the size of a small egg which suggested another cyst, but as there was no fluid on

Fig. 4.—About two and one-half months after operation. The cyst contains a small quantity of barium suspension which is shown in the dependent position. The lung below the cyst is becoming clear. The rubber drain is transparent and does not show. On fluoroscopy, in two directions with a large probe passed through the tube, the presence of the latter within the cyst was fully established. There is some contraction of the intercostal spaces but the cyst wall does not collapse. March 9, 1928.

aspiration and the patient's condition was precarious, I did not follow up this matter. It had not appeared as a cyst in the x-ray pictures, and may have been a pulmonary condition of some other variety but probably congenital. Free incision was now made in the bottom of the cyst, and a piece of its wall, tough and evidently embryonic skin, was excised for laboratory examination. On entering the cavity, a large amount of pus was discharged. It had to be removed by wiping and suction. Two soft black rubber tubes each the size of an adult middle finger were then placed within the cyst and led out of the anterior part of the wound so as to bring the contemplated permanent opening in a location which could be reached by the patient herself. In order to prevent this from

closing, pieces of the eighth and ninth ribs were resected together with their periosteum. Packings of gauze were then placed alongside the tubes and the chest closed with pericostal sutures of chromicized catgut. Other sutures approximated the muscle planes, but the skin was merely packed with a narrow strip of gauze. This operation was followed by dyspnea, later relieved by burying one of the tubes beneath the skin while the other was fitted with a finger-cot valve [1] The dyspnea at once disappeared and in a few days the patient was out of bed. The formation of a fistula by granulation was extremely slow so that about two weeks after the operation, when I removed the second buried drainage tube, examination of the pleura did not reveal granulation tissue and healing reaction seemed absent. The pericardium was clearly visible through its covering of thin pleura. The cyst was now entirely empty but not collapsed.

Finally, an organized canal did form in the tract of the tube between the cyst and the outer world. So long as the patient wears the valve there is no respira-

Fig. 5.—Lateral view of the patient same day as figure 4. The barium suspension is again seen. The dimensions of the cyst are displayed in this new direction.

tory embarrassment and she is comfortable leading her normal life, but when the valve is omitted, even during the dressings, there is still (now four months after operation) sufficient mediastinal mobility to produce this disagreeable symptom.

The discharge became mucoid and slight amounting only to about one-half ounce a day. It is hoped that even this may diminish. There has been a great gain in weight and sense of well-being.

The tube has been replaced by one of smaller caliber, about 40 F., but it is expected that even though an epithelialized lining forms between the outer skin and the lining of the cyst, it will be necessary for the patient to wear a tube.

Whether with the gradual stiffening of the mediastinum the valve will still be indispensable cannot be foretold, but an advantage in continuing it for a long

1. This valve is a modification of a valve devised by Thiersch many years ago. and made airtight with adhesive strapping of the skin.

time would be its tendency to displace the mediastinum toward the pathologic side by suction, the space being eventually filled or partly filled by the physiologic emphysema of the contralateral healthy lung.

Owing to the patient's age, it is not considered wise to make any effort to extirpate the wall of the cyst or even to collapse the chest on that side by thoracoplasty.

The diagnosis of cyst of the lung in this case is made partly by exclusion and partly because of the mucoid character of the secretion

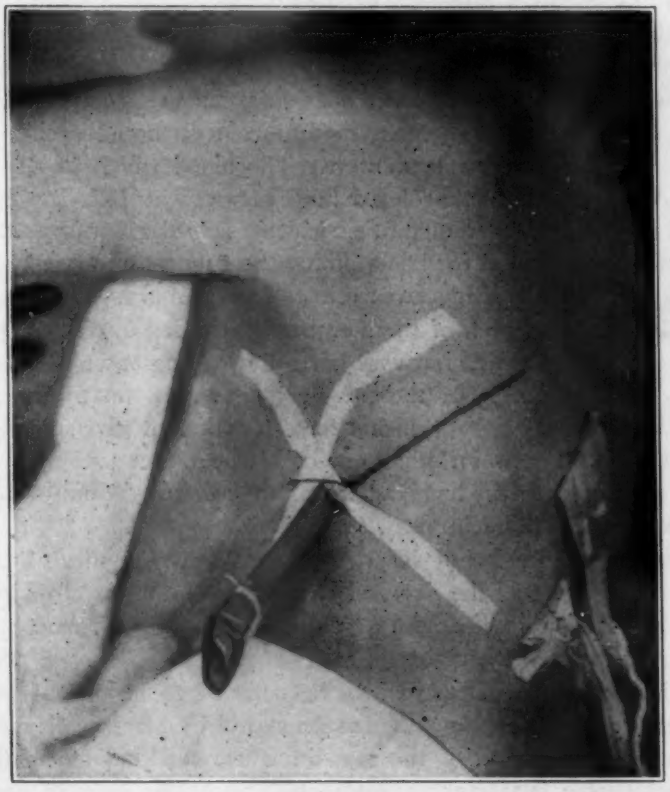

Fig. 6.—Picture taken in the office, the patient having traveled alone from a neighboring city to New York. March 9, 1928. Note the tube and finger-cot valve.

from its walls when the blood and pus following the operation had ceased and no longer confused the picture. There is a great globular mass within the lung, its walls containing large vessels. The tumor contents did not show any evidence of material such as is invariably found in dermoid cysts or teratomas. There never was any cough or expectoration. The piece of wall of the cyst was described by Dr. Louis Gross, Director of Laboratories at Mount Sinai Hospital, in the following words:

"Cyst wall of hyaline connective tissue. The lung tissue in the vicinity of this cyst wall shows organization and dilated bronchi. No bacteria found."

The description of the hyaline tissue is somewhat in line with that of the congenital pulmonary cyst wall described by Sydney Smith, Principal Medico-Legal Expert, Egyptian Government,[2] in reporting a case of multiple cysts in a new-born child. He stated that "They were smooth walled and lined in certain cases with flattened cells. There was no cubical epithelium lining the cavities nor muscular or elastic fibres in the walls. Here and there the cavities were filled with mucoid-looking or colloid-looking material."

Although epithelial cells were not found in the specimen which was given to Dr. Gross, yet the continued mucoid discharge would indicate the presence of secreting membrane. The blood which was found on aspiration in the chocolate-colored fluid removed at Jefferson Hospital as well as in the larger quantities which escaped on my opening the cyst could be accounted for by assuming the rupture of vessels with the increasing tension resulting from the stretching of the walls of the sac.

An incomplete search of the literature has failed to disclose the history of any case in which recovery has followed operation. Most of the patients were infants, but there were adults even up to the age of 68 years. The longest list which I have seen is that given by Koontz.[3] In his list of 108 cases including one of his own, there are many which are not pure cysts of the lung but which are congenital deformities such as bronchiectases, blind bronchi, etc.

Since the publication of Koontz' paper I have found an article by Robert T. Miller from the Surgical Department of Johns Hopkins University and Hospital.[4] In both of these papers will be found a wealth of material to which the reader is referred.

Neuhof [5] reported a case which he believed to be one of infected congenital cyst of the lung. It was improved by marsupialization. A microscopic examination of the cyst lining revealed ciliated epithelium of bronchiogenic character. This would place this case in the class of those produced by the pinching off of a bronchiole. Aspiration at the time of operation withdrew sterile pus.

From my perusal of various contributions on this subject, I conclude that there are possibly two principal causes for the production of this condition; first, an embryonic pinching off of a bronchus, bronchiole or

2. Smith, Sydney: Brit. M. J. **1**:1005 (May 30) 1925.

3. Koontz, A. R.: Bull. Johns Hopkins Hosp. **37**:340 (Nov.) 1925.

4. Miller, Robert T.: Congenital Cystic Lung, Arch. Surg. **12**:392 (Jan.) 1926.

5. Neuhof: Ann. Surg. **87**:606 (April) 1928.

smaller air passage with the formation of a retention cyst no longer connected with the open bronchial tree and, second, represented by the opinion of Grawitz, that the cysts may be due to faulty anlages of the lymph vessel systems of the corresponding lung. To repeat in the simplest possible words, one type is of bronchiogenic origin, the other of interstitial origin.

The case which I report may be of either type judging by the histologic appearances. With the total absence of epithelium and with no typical organized structure, I may well be dealing with a case of the interstitial type. If at some future time it should be feasible to remove by curet or otherwise a small specimen of the lining of the wall of the cyst, the presence of almost any kind of epithelium, cuboid, cylindrical or flat, with or without cilia, may assign the case to the bronchiogenic or bronchiectatic variety. Had this patient been younger or more robust I should not be satisfied with the mere formation of this permanent fistula even though the result were comparatively satisfactory, but would perhaps make an attempt to extirpate or to collapse. Probably the most important single factor leading to recovery in this instance was the employment of the valve tube which eliminated the distress of mediastinal commotion, the greatest danger of all.

ROENTGENOLOGIC DIAGNOSIS OF THORACIC DERMOIDS

LEON THEODORE LE WALD, M.D.
Professor of Roentgenology, New York University and Bellevue Hospital Medical College and Attending Roentgenologist, Willard Parker Hospital
NEW YORK

In the course of routine roentgenologic examinations of the chest I have observed a number of cases of intrathoracic dermoids, and the information gained in this series of cases will probably be of interest.

The term dermoid is used in the title rather than teratoma, because the majority of the cases observed have been benign. However, in spite of the fact that the dermoids were benign, they became dangerous to life in some cases on account of their size.

In some instances the tumors, closely associated with the mediastinum, offered considerable difficulty in differential diagnosis, especially in differentiation from aneurysm. In this connection I would state that the usually accepted statement in the textbooks that on fluoroscopic examination the determination of visible pulsation in a mediastinal tumor is a simple matter of observation and a positive means of diagnosis between aneurysm and tumor is by no means always true. This fact should be given careful consideration so that those inexperienced in the use of the fluoroscope may be placed on guard in order that an error in diagnosis may be avoided. For example, in case 2 (fig. 3), which was presented at a meeting of the New York Roentgen Society, a number of experienced roentgenologists were asked to express an opinion as to the presence or absence of expansile pulsation. About an equal number of those present stated that in their opinion expansile pulsation was present, while the others expressed the opposite view that the appearance was due to transmitted pulsation from the heart or aorta. In this case operation was successfully performed by Dr. Howard Lilienthal at Bellevue Hospital, and the condition was proved to be a dermoid of the mediastinum.

Dr. William C. Lusk, at the time when he was wiring aneurysms as a routine, told me that one of his greatest problems was to be sure that a given case was one of true aneurysm and not of mediastinal tumor (fig. 1). He said that he had rejected a number of patients who had journeyed from distant places to have the wiring operation performed, only to be told that they were not suffering from aneurysm.

The differential diagnosis between intrathoracic dermoid and a primary or secondary malignant growth is sometimes made with difficulty. The slowness of growth is at times the only available means at the disposal of the roentgenologist in making the differential diagnosis. In some instances, exploratory thoracotomy would be a justifiable procedure, as in this way an early malignant tumor might be successfully removed; if the case proved to be one of dermoid, no harm would have resulted from the operation under ordinary circumstances, and at the same time the dermoid cyst might have been successfully dealt with.

At times a teratoma may be multiple. One case of this sort was encountered.

The following cases were roentgenologically examined by me, and have been previously reported from the clinical and pathologic standpoint.[1]

REPORT OF CASES

CASE 1.—J. K., married, aged 37, a plasterer, was born in Russia. He came to the clinic of the New York University and Bellevue Hospital Medical College on July 19, 1917. He said that he had been ill for five months with pain in his chest. On further questioning, he said that he had pains in his chest for twenty years. Physical examination made by Dr. Margaretten disclosed dulness below the left clavicle. The Wassermann reaction of the blood was negative. An x-ray examination made by me disclosed a mass in the left side of the chest measuring about 8 by 12 cm. in diameter, extending from the heart shadow toward the lateral wall of the chest, and about equal in density with the heart shadow (fig. 1). The edges were smooth, and no expansile pulsation was observed. The man was observed for about one year and no change in the size of the intrathoracic mass was seen.

This patient came under observation in the Presbyterian Hospital on Dec. 18, 1918, and the subsequent history, as given in the hospital, revealed the following: On admission, the temperature was 102.8 F.; pulse rate, 106 and respirations, 46. The chief complaint was a pain in the chest of thirty-six hours' duration. The present illness was sudden in onset, with sharp, severe, pleuritic pain, dyspnea, chill, fever, cough, malaise and prostration and bloody expectoration. Physical examination showed that breathing was somewhat labored. There was a rasping frequent cough, which was productive. Over the left lung anteriorly, there were subcrepitant râles; there was no diminution in resonance of percussion note, and the fremitus was normal. Over the left lung posteriorly, there was diminished resonance on percussion, markedly diminished breath sounds and quantities of moist râles.

On December 27, at 4 p. m., there was a distinct rise in temperature and pulse rate. The temperature was 101.8 F.; respirations, 20, and pulse rate, 80. At 4:45 p. m., the patient coughed up from 6 to 8 ounces (178 to 236 cc.) of greenish, thick, very foul smelling fluid.

On December 28, over the left anterior side of the chest in the second and third spaces, there were signs which suggested a cavity.

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1. Lambert, S. M., and Knox, L. C.: Intrathoracic Teratoma, Tr. A. Am. Phys., 1920.

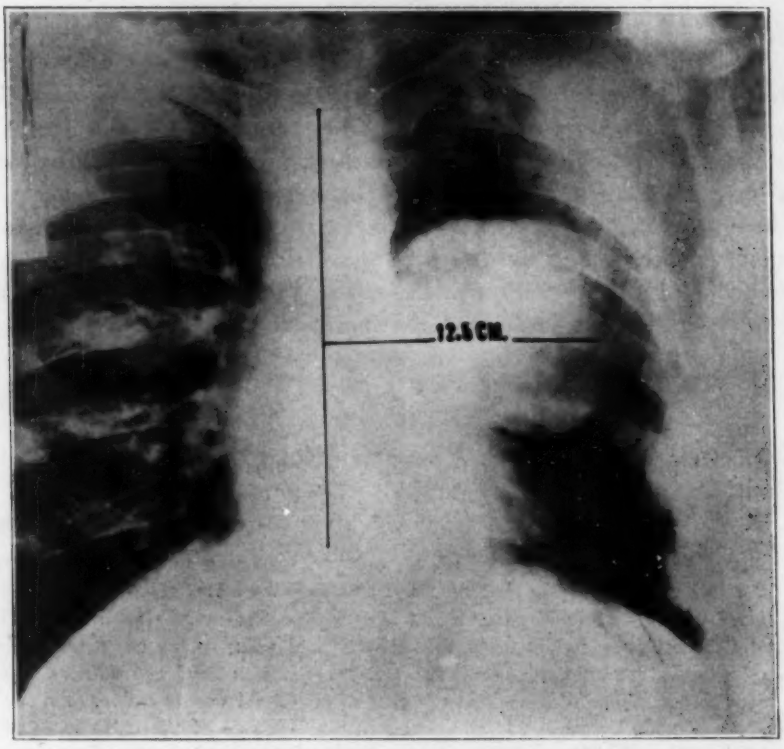

Fig. 1 (case 1).—Mediastinal dermoid. The shadow somewhat simulates aneurysm of the aorta; note that it disappeared after spontaneous evacuation through a bronchus two years later (fig. 2 *B*).

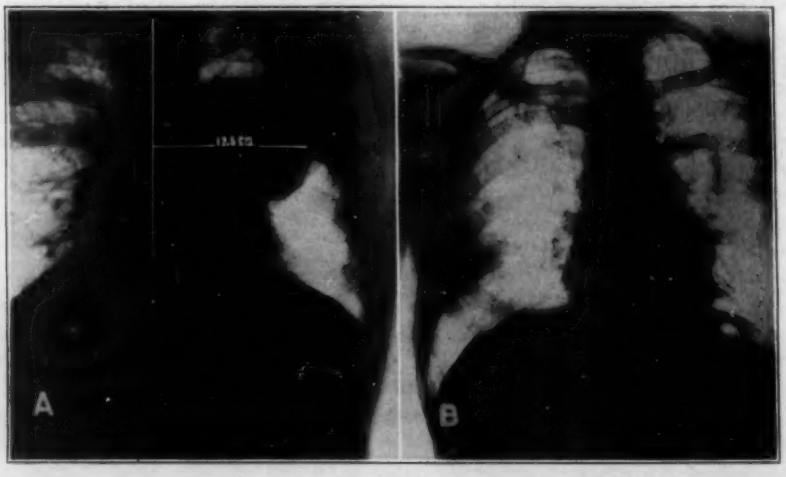

Fig. 2 (case 1).—*A* shows a mediastinal dermoid; *B*, after spontaneous evacuation of the contents through a bronchus two years later.

The fever disappeared slowly, and the patient made a good convalescence. On Jan. 7, 1919, the lungs were practically clear of signs. A letter from the patient written on May 15, 1920, stated that he felt well, was able to work and considered himself cured.

Figure 1 shows a distinct tumor mass in the root of the left lung as existing on July 19, 1917, and a second radiograph made fourteen months later, Sept. 13, 1918, showed the same condition. Three months later, after an attack of fever, the patient coughed up a large amount of material. X-ray examination the next day showed a distinct cavity at the location in the chest of the tumor mass.

The diagnosis of dermoid cyst, which became infected and ruptured into a bronchus, seemed justified by the comparative study of the roentgenograms (fig. 2).

CASE 2.—J. W., white, aged 19, single, a laborer, came to the clinic of the New York University and Bellevue Hospital Medical College, on Jan. 25, 1916,

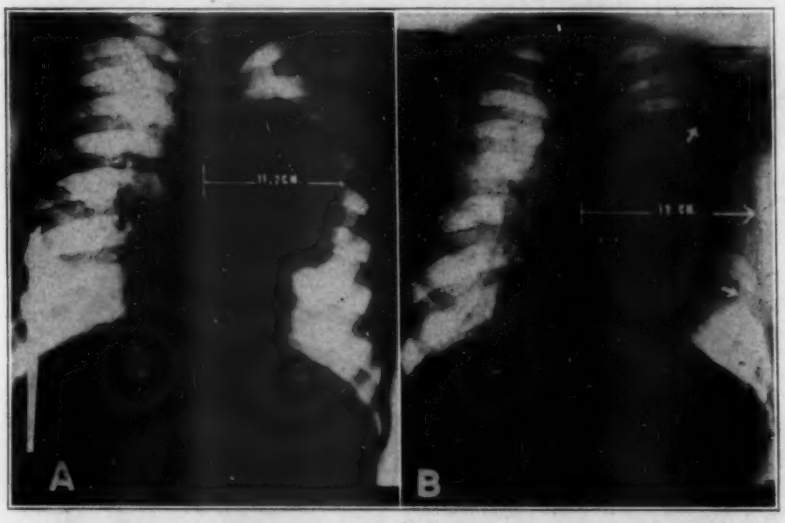

Fig. 3 (case 2).—Mediastinal dermoid. *A* shows the condition when the patient was first examined; *B*, three years later. Note the increased size of the shadow. X-ray diagnosis was confirmed by operation (Lilienthal) nine years later.

complaining of headaches. He said that he had had headaches intermittently for a period of two years, which had gradually increased in severity. In the course of a routine physical examination made by Dr. E. J. Riley, it was discovered there was dulness over the left side of the chest, with obscure physical signs. The Wassermann examination was negative.

Radiographic examination showed a shadow to the left of the median line and above the left auricular shadow. It extended about 9 cm. to the left of the median line and about 10 cm. in vertical diameter. The edges were rounded and the lower border was superimposed on that of the left ventricle. The shadow was as dense as the heart shadow. On fluoroscopic examination, the portion of the mass near the heart seemed to show some movement, but it was probably transmitted from the heart. No definite expansile pulsation was made out. On Nov. 16, 1916, a second examination was made which showed a slight increase in the dimensions

of the tumor of the chest. A third examination on March 5, 1919, showed a marked increase in the size of the tumor. It had reached nearer to the surface of the chest, having increased about 3.3 cm. in all directions. The heart was pushed to the right. It would seem that this was a case of true dermoid of the anterior mediastinum. Figure 3 *A* represents the radiograph made in January, 1916, and figure 3 *B* that made in March, 1919. The gradual growth of the tumor is clearly demonstrated.

This patient was recently operated on in Bellevue Hospital by Dr. Howard Lilienthal, who made a final diagnosis of mediastinal dermoid. Dr. Lilienthal[2] informed me that the patient is making an excellent recovery, although a fistula is present.

CASE 3.—P. L., a hod carrier, aged 40, married, was first seen on April 14, 1916. This patient is from the service of Dr. Samuel W. Lambert at St. Luke's Hospital. I am indebted to him for the clinical record. According to the past history, he was stabbed in the chest twenty-two years before. He spat blood

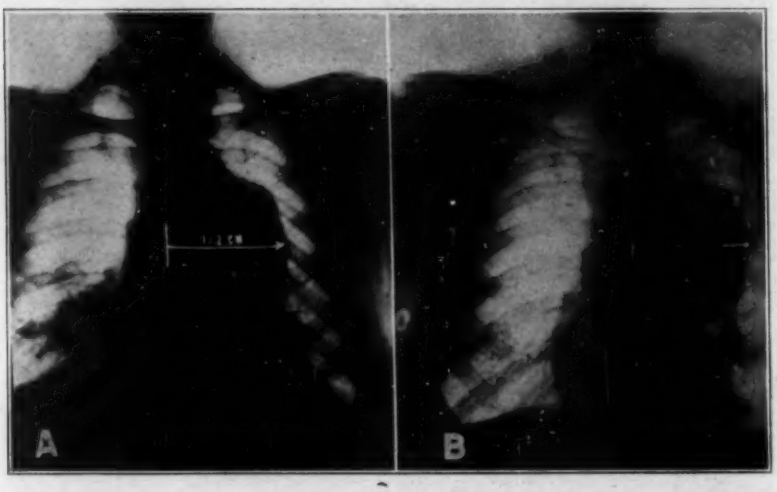

Fig. 4 (case 3).—Mediastinal dermoid. *A* shows the condition when the patient was first examined; *B*, two years later. See figure 5 for the lateral view showing erosion of vertebrae.

at the time of the injury and was sick one week. He admitted gonococcic and doubtful syphilitic infection. He used alcohol moderately. He had been in the United States for thirteen years. The present history showed that he had constant pain in the left side of the dorsal region. There were no cough, sweats, dyspnea, bladder symptoms, palpitation, hemoptysis nor dysphagia.

Physical examination showed a well nourished man, with good color, weighing 150 pounds (68 Kg.). There was no change in the breath and voice sounds and no râles were heard; there was dulness to the left of the sternum from the second rib downward continuous with the cardiac dulness and extending to the left for 3 inches (7.6 cm.) from the midline of the sternum. The heart was not enlarged; no murmurs were heard in the heart or over the area of dulness on the anterior wall of the chest. The spine did not show tenderness or irregularities and there

2. Personal communication.

was no kyphosis. There was a scar on the back of the right side of the chest in the scapular line over the ninth rib. The Wassermann reaction was negative (April 22, 1916). The blood pressure was 126 systolic, 76 diastolic, and equal in both arms.

Radiographic examination (fig. 4) showed a rounded shadow extending above and to the left of the heart, 13.5 cm. from the midline of the body, which did not pulsate and which had a broad (6 cm.) extension downward in the midline behind

Fig. 5.—Lateral view showing erosion of the bodies of the vertebrae from pressure of the mediastinal dermoid (fig. 4). The patient was living eight years later. The anterior view is shown in figure 4.

the heart and reaching to the dome of the diaphragm. A side view showed the anteroposterior contours of the tumor to be also globular and situated behind the arch of the aorta and the heart. The posterior aspect of the mass fitted into a sort of bed on the anterior surfaces of the four of the vertebrae, which were deformed in a symmetrical curve. It would seem that this development occurred during growth and that the spinal bones had been prevented from attaining their

normal shape by the pressure of the neoplasm. This deformity presented a smooth even surface and in no sense could it be considered as an erosion or an inflammatory process (fig. 5).

The patient has remained in good health except for recurring mild attacks of pain. He returned to work and carried a hod still in April, 1920. On April 11, 1920, the physical signs were unchanged from those four years before and a radiographic study of his thorax did not show any changes from those originally found and already described. The original diagnosis of dermoid in the posterior mediastinum seemed justified by the presence of a large, smooth, globular tumor behind the heart, which had apparently caused by its mere presence a developmental failure to grow normally in the bodies of the fourth, fifth, sixth and seventh dorsal vertebrae, and had compelled these bones to form a concave surface on their ventral borders closely applied to the posterior surface of the tumor.

This diagnosis was substantiated by an entire lack of growth in the tumor during the four years of observation, and by the possibility of not showing pulsation in the mass by x-ray examination, especially from the lateral view, and of separating the tumor from the arch of the aorta lying in front of it. The absences of pulsation, thrill, murmurs, tracheal tug and of radial pulse variation were also confirmatory, although the roentgenogram taken from the front showed the tumor in the possible location of an aortic aneurysm.

This patient, after a period of twelve years, is reported to be alive and in Rochester, New York, and roentgenograms now show little change from the condition shown in figure 4.

CONCLUSIONS

1. Intrathoracic dermoids are much more frequent than was believed prior to routine x-ray examination of the chest.

2. The early roentgenologic recognition of the presence of a dermoid tumor in the chest renders it possible for the thoracic surgeon to deal successfully with the lesion surgically before complications have occurred, such as pressure on a bronchus resulting in retained bronchial secretions, or abscess of the lung.

3. The differential diagnosis between dermoid tumors and other lesions can be successfully made in many cases by repeated roentgenologic examinations in various positions, especially in the direct lateral, with an interval of several weeks or months between examinations in doubtful cases.

4. Roentgenologic examination of the chest of every person should be made in early life and repeated at intervals in order to recognize the presence of a dermoid tumor or other pulmonary or mediastinal lesion at the earliest possible time, so that the question of its advance may be accurately determined and properly treated.

PRIMARY CANCER OF THE LUNG

WILLY MEYER, M.D.
NEW YORK

The most tormenting types of advanced cancer known in medical science are those of the tubes through which food and oxygen—the two factors that sustain life—are introduced into the system, cancer of the esophagus and cancer of the bronchial tree.

In advanced cancer of the esophagus, gastrostomy as a palliative operation opens a by-path for the introduction of food and brings to the affected area rest and the elimination of irritation, although the swallowing of the ever reaccumulating saliva down to, or past, the obstructing tumor, remains as a source of constant annoyance to the patient.

But in advanced cancer of the bronchial tree with its usual intrapulmonary suppuration there is no chance of creating a substitute entrance for the ingress and egress of the air. The rate of respiration is from twelve to eighteen times a minute and causes irritation which results in cough and expectoration The patient is never at rest, except when placed under the influence of drugs. Cancer of the lung, therefore, is a much more cruel foe to mankind than that of the esophagus.

REPORT OF A CASE

The following recently observed case of cancer of the lung has prompted me to write this paper.

A man, aged 66, formerly a chemist for many years, had always been well until September, 1927, when he developed a cough which persisted in spite of treatment. The origin of the cough remained obscure but influenza was thought of as the immediate cause. The cough tormented the patient day and night. The sputum was mucopurulent, with a trace of blood at one time. A slight hoarseness developed. Over the upper and middle lobes of the right lung increasing flatness with bronchial breathing was noted. There was a slow but steady increase of symptoms; the patient became bedridden and lost weight. A bedside roentgenogram showed a dense shadow over the affected region.

At this stage, in February, 1928, the patient was transferred to the Lenox Hill Hospital, where he came under my care. An attempt was made to establish a definite diagnosis in the usual systematic way. The conditions considered in the differential diagnosis were: abscess, sacculated empyema, tumor, with or without subacute and chronic pneumonitis. On examination, the epigastric anastomosing veins on the right side were found to be markedly distended; there was also slight edema of the right arm. The previously found absolute flatness over the upper and middle lobes of the right lung was noted, and sharp, bronchial breathing with a few râles was heard. Pectoral fremitus was much reduced over the affected area; voice, breath, and whispered sounds were markedly increased. The patient had a deep,

rough, hoarse voice, and occasionally had difficulty in swallowing. The pulse was weak and intermittent. The temperature was slightly elevated. The laboratory examinations showed: slight secondary anemia; white blood corpuscles, 14,200; polymorphonuclears, 83 per cent; no cellular malignant elements in the sputum, the culture of which revealed pneumococcus, nonhemolytic streptococcus, *Staphylococcus albus* and *Micrococcus siccus*. The Wassermann reaction was negative. The urine showed hyaline and granular casts with albumin.

X-ray examination showed that the entire upper lobe of the right lung was involved in a dense shadow without aeration. Extending out from the right root into the middle lobe, there was also some density (figs. 1, 2 and 3). The lower

Fig. 1.—Patient with cancer of the lung, in prone position.

lobe was apparently well aerated, although not much of this portion of the lung could be seen on account of the height of the right side of the diaphragm (fig. 2). The costophrenic angle was clear. On the left side, there was compensatory aeration. The heart and mediastinal shadows seemed to be displaced somewhat to the left, but it was possible that the position of the patient may have had something to do with this apparent displacement.

On further study of the x-ray pictures, it appeared that the two halves of the diaphragm were at different levels with the patient in the horizontal (fig. 1) and in the erect posture (fig. 2). The first day after the patient's admission to the hospital, the radiologist did not dare let him sit up for any length of time. In the recumbent posture, both halves of the diaphragm appeared to be almost on the same level. But in the erect posture on the following day, the diaphragm on the

Fig. 2.—Same patient in erect posture.

Fig. 3.—Same patient in lateral posture.

right side stood fully 1½ inches (3.7 cm.) higher, evidently due to the complete chronic atelectasis of the upper and middle lobe.

As bedside x-ray pictures of older pneumonic persons are usually taken with the patient lying on his back, the foregoing observation, if seen in a series of cases, may be of some value when drawing conclusions from the position of the diaphragm.

All the roentgenographic observations pointed toward a tumor, probably bronchial, which was causing complete obstruction of the upper main bronchus and partial obstruction of the main bronchus of the middle lobe. There was no roentgenographic evidence of bronchiectatic cavities.

On the basis of these observations abscess of the lung and sacculated empyema were ruled out of the diagnosis. The clinical picture was that of an advanced cancer of the lung with the usual acute and subacute lobular pneumonitis and beginning bronchiectasis, having originated probably in the main bronchus and now obstructing the bronchus of the upper lobe, and probably also the bronchus of the middle lobe of the right lung. The absolute flatness noted, with the x-ray observations and high elevation of the right part of the diaphragm, indicated massive chronic atelectasis. Bronchoscopy, though desirable for corroboration, as well as laryngoscopy, appeared contraindicated, at least for the present, in view of the patient's condition. Operative intervention of any kind was out of the question. Had there been the usual severe neuritic disturbances so frequently seen in tumors of the anterior mediastinum, there might have been an indication for a palliative operation on the bony structure of the thorax under local anesthesia for the purpose of decompression.

Physical therapy was instituted, deep x-ray treatment, diathermy and alpine sun being used. A few séances could be given. On the twelfth day of the patient's stay at the hospital, death occurred suddenly. Autopsy was granted. The principal observations were: carcinoma of the right upper (eparterial) bronchus with infiltration of the upper lobe of the right lung, trachea and right main bronchus; metastases in tracheal, bronchial and mediastinal lymph nodes, compressing the esophagus, aortic arch and right pneumogastric nerve; purulent bronchitis and bronchiectasis of the upper lobe of the right lung with organizing pneumonia of the upper and middle lobes and acute lobular pneumonia of the lower lobe of that lung. The microscopic diagnosis was alveolar carcinoma.

The cause of the development of cancer of the lung is a chronic irritation as in every other case of cancer. The chronic irritation may be due to the breathing of the gaseous distillation products of tar from the exhaust of automobiles in the streets, the inhaling of cigaret smoke (?), and the inhalation of minute particles of dust on streets and roads and of ashes, soot, chemicals, etc., floating in the air, particularly in and near factories, also in certain mines (Schneeberger Lungenkrebs). Influenza, pneumonia and chronic bronchitis may also furnish the chronic irritation which in the course of time induces the development of cancer in persons who are predisposed to the disease.

Recent statistics would seem to indicate that primary cancer of the lung is increasing, the incidence keeping pace with the increasing number of opportunities for the development of the disease.

The fact that in from 85 to 90 per cent of the cases the disease develops primarily in the larger bronchi and in their immediate subdivisions and not in the parenchyma of the lung is probably ascribable to physical causes. The bronchi are at rest; the parenchyma is in continuous motion and has a most active circulation. There is a much more active circulation of the blood in the alveoli where the exchange of gases takes place. The view that cancer is not likely to arise in any locality where there is active circulation of the blood is supported by numerous instances. One need but refer to the rareness of cancer of the heart, of the duodenum, of the cross-striped muscles, and of the much abused breast of the milk cow on the farm.

In the rare cases of cancer of the lung at the periphery of the organ, the original tumor probably also started in one of the smaller bronchi, not in the alveoli.

Cancer is more often found in the right than in the left bronchus. The reason for this is most likely the anatomic fact that the right main bronchus is larger and descends more perpendicularly than the left. Both factors, it is assumed, contribute to the more ready entrance of irritating causes on the right side. These causes, as it seems, affect primarily the circular glands of the bronchus.

The foregoing advanced case of cancer of the lung with its intense and continuous suffering and my utter inability to relieve the condition, gave me an additional strong impetus to ponder the question as to what might be done to prevent these patients from reaching such a deplorable stage.

In the beginning, a malignant tumor in one of the bronchi probably acts like a foreign body of small size in that it constitutes a source of recurring irritation followed by incessant cough, for which there is no etiologic explanation, such as influenza, pneumonia, etc. In the light of the evolution of modern medical science, such cases are considered borderline cases as there is cause for cancer to be suspected. The medical man should insist on an immediate x-ray examination, and careful analysis of the sputum and of the blood; if the results are negative, he should call in a trained bronchoscopist. The latter will often detect an infiltration or elevation in one of the main bronchi or in their first or second divisions, and, if feasible, he will make a biopsy. The microscope will then shed the desired light on the situation, and a definite diagnosis can be made.

But the case should not rest here. Aggressive intrabronchial therapy should follow promptly. With a properly constructed galvanocautery or by means of endothermy, the specialist should thoroughly destroy the diagnosed source of the trouble and superficially cauterize its immediately adjacent area. He should then watch developments at regular

intervals and do what to him seems best under the circumstances. Should any suspicious spot reappear within the bronchus, he should repeat the cauterization or refer the patient to the surgeon.

The treatment of patients with incipient cancer of the bronchus, it seems to me, clearly belongs to the domain of the bronchoscopist. This may seem to some but a fantastic dream of the future. Yet, it may become a practical reality at once if in these cases with a dubious diagnosis the internist learns to cooperate with a trained bronchoscopist, as the surgeon has learned to do in the course of the last ten to fifteen years. In fact, the "dream" has already become a reality, as proved by a recent case reported by Dr. John D. Kernan of New York before the New York Society for Thoracic Surgery on March 27, 1928.[1]

A case perhaps belonging to this class was reported in a personal communication to me in 1922.[2] It referred to an intrabronchial endothelioma removed by means of bronchoscopy by Chevalier Jackson of Philadelphia. The tumor had caused suppuration. Dr. Jackson added, "Inasmuch as the man is alive and well six years after the operation the growth has to be considered benign, though histologically malignant."

Patients with peripheral cancer of the lung should be treated by the surgeon. The earlier the patient comes to operation, the better. Cauterylobectomy will probably prove to be the operation of choice.

As has so often been emphasized by me, surgery and bronchoscopy are the closest allies in the treatment of diseases of the lung and of the esophagus. The expert peroral endoscopist today is the best friend of the surgeon, and still more so of the patient. It is most desirable that the peroral endoscopist should also become the close ally and friend of the internist!

In 1922, I said: "Who can tell but that perhaps in the future the medical man will join hands with the bronchoscopist, even in the treatment of pneumonia proper, not alone of the aspirating type, when it comes to particularly difficult, long drawn out cases."[2] "It will need courage," I added, "but if once started, and perhaps if it brings relief in certain difficult cases, the foundation for this team work will have been laid."[3] Meanwhile, this courage has been exhibited in the highest degree at two of the principal hospitals of New York. The bronchoscopist was called in by the internist to treat with him not only patients with "particularly difficult and long drawn out cases" of pneumonitis, but patients with cases of acute lobar pneumonia, the indication for such a procedure having been found on the basis of most inspiring results

1. Kernan, John D.: Carcinoma of the Lung: Report of a Case, Arch. Surg., to be published in this issue.

2. Meyer, Willy: Observations of Lung Suppuration and Its Treatment, Arch. Surg. **6**:361 (Jan.) 1923.

3. Meyer (footnote 2, pp. 369 and 410).

obtained by bronchoscopy in animal experimentation.[4] While this is still an isolated step, the results of which are eagerly watched by a large number of medical men, it is nevertheless a start.

When such inspiring teamwork between internist and bronchoscopist is brought about more generally, I have no doubt that cancer of the lung, or better, cancer of the bronchi, will cease to show a mortality of 100 per cent, as it does up to the present time.

It is deplorable that this condition still exists in spite of the fact that surgical procedures on the thorax have been widely and safely used for the last ten to fifteen years. But this deplorable condition is not due to any lack of energy on the part of the surgeon, who in the end is called on to treat the patient, but to the lack of cooperation already mentioned. In almost every instance the disease has been far advanced when these patients have come to the man who could perhaps, or probably, have given radical help had he been consulted earlier. Thus far these cases have always reached the modern surgeon too late to make it feasible for him to practice progressive surgical measures, namely, to expose the bifurcation of the trachea; open and inspect the inner surface of the tumor-bearing portion of the bronchus and destroy the growth by cautery or by means of endothermy under the direct guidance of his eyes, then close the bronchus with sutures; or, in cases that appear to need more radical measures, open, inspect and resect the tumor-bearing portion of the bronchus; crush, invert and close by suture the proximal stump and stitch the distant end into the chest wound, thus preserving the function of the lung proper. In every patient who has come under my care up to the present time, the tumor had grown into the parenchyma of the lung, and the respective lymph nodes were extensively involved, thus frustrating every hope of a cure. With proper cooperation by the internist, the bronchoscopist could refer to the surgeon patients with bronchial malignant infiltration, who are seen early, but whose trouble fails to yield to intrabronchial treatment, and the surgeon might then still be able to give radical help, in a certain percentage of cases at least.

The future progress here, as in malignant diseases of the esophagus, clearly lies in early diagnosis and early treatment. As the public has learned to proceed in acute appendicitis, namely, to ask promptly for a definite diagnosis and immediate surgical treatment, it should be taught to act in cases of unexplainable cough as well as in cases of beginning difficulty in swallowing. The agencies for the medical education of the public will find a useful extension of their beneficial work in urging these symptoms on the attention of the public. People must learn that tem-

4. Coryllos, Pol N.: Lobar Pneumonia a Pneumococcic Massive Atelectasis: Suggestion of Bronchoscopic Treatment, to be published in this issue.

porizing in cases of this nature causes them to lose the only chance they have of saving their lives.

When this shall have been accomplished and when in case of an unexplained, persistent cough and of beginning difficulty in swallowing, the internist calls in the trained peroral endoscopist, and the endoscopist calls in the specialist in treatment by irradiation and the surgeon, should he need them, the cure of patients with cancer of the lung and cancer of the esophagus will be heard of oftener.

Cooperation, the teamwork between medicine and surgery and the various specialties of our science, is bound to bring about the improved results so urgently needed in the treatment of this class of patients.

CARCINOMA OF THE LUNG

JOHN D. KERNAN, M.D.
AND
ARTHUR J. CRACOVANER, M.D.
NEW YORK

In a survey of the literature of the past six years on carcinoma of the lung, a great many articles were found dealing with its increasing frequency, and giving detailed statistical studies. There is also considerable literature concerning the possible etiology, the pathology and diagnosis of carcinoma of the lung, but little is mentioned about treatment, probably because few cases have been dealt with successfully.

Surgical treatment is recommended by a number of men, but few successful cases are reported (Sauerbruch, Davies, Lilienthal, Lenhartz). Roentgen therapy has been attempted, but with the fear that fibrosis of the lung may accompany the shrinkage of the tumor, as it sometimes has done (Rolland, Schroeder). This therapy is perhaps used as a palliative measure, with the hope of some temporary relief. Radium has also been applied externally, but without success.

There is still another manner of treatment which has been mentioned only occasionally. Many authors have readily acknowledged the remarkable advance and the possibility of making an early and more certain diagnosis of carcinoma of the lung by means of bronchoscopy. However, the possibility of treatment has not been generally recognized. In 1921, Yankauer had a patient who was treated by implantation of radium seeds through the bronchoscope directly into the tumor. He also applied radium externally. In 1923, D. C. Greene presented a case of malignant tumor of the bronchus of doubtful type. There was collapse of the lung in this case. Excision was performed; radium was applied through the bronchoscope, and deep roentgen therapy was used with some success. The case was reported a short time after treatment had been given. H. B. Orton excised through the bronchoscope a bronchial papillary growth reported malignant. This case was reported about four months after treatment was instituted. Kully merely mentioned that radium therapy could be made more effective by introducing the radium capsules directly into the growth through the bronchoscope.

We shall report a case of carcinoma situated in the left main bronchus, which caused an intermittent complete atelectasis of the lung. Treatment consisted of the insertion of radium needles, and surgical diathermy administered through the bronchoscope.

Fig. 1.—Roentgenogram taken during attack on Dec. 28, 1926.

Fig. 2.—Roentgenogram taken after injection of iodized oil 40 per cent on Jan. 21, 1927.

REPORT OF A CASE

A. B., white, a woman, married, referred by Dr. Fred Henry Mosler, gave a past history that was negative, except for attacks of hay-fever each year for the last fifteen years. In December, 1922, the patient complained of a heavy sensation in the chest and dyspnea. In September, 1923, she had another attack in which she had much more marked dyspnea. The diagnosis at that time was asthma. During 1924, she was apparently well. In 1925, she began to have difficulty in breathing again, and in September of that year she suffered severe pain in the left side of her chest and marked dyspnea. The diagnosis at this time was collapse of the left lung. This was confirmed by x-ray examination. The results of bronchoscopy at this time were reported essentially negative except for a doubtful slight constriction. This attack lasted two weeks, when the lung again expanded, and breath sounds could be heard over the entire chest. Four weeks later, the results of the x-ray and fluoroscopic examinations were negative. In February, 1926, the patient had another attack, diagnosed as pleuropneumonia. She had fever at this time. The x-ray picture revealed a collapse of the left lung. Eight weeks later, the x-ray picture was again relatively normal. From April to July, 1926, the patient gained 7 pounds (3.2 Kg.) in weight, and was feeling well again. In December, 1926, the present attack began.

The patient was admitted to the Lenox Hill Hospital on Dec. 13, 1926. She was complaining of marked dyspnea and pain in the left side of the chest. She appeared acutely ill, with a temperature of 103.4 F.; a pulse rate of 114, and respirations, 28. She was cyanotic and very uncomfortable, with a distressing dyspnea.

The physical signs gave the impression of solidarity of the left lung. She was observed for a few days. Her temperature remained high, going up to 104.4 F. She coughed considerably and expectorated a moderate amount of phlegm. X-ray examinations were unsatisfactory, and reports were doubtful at this time. A provisional diagnosis of atelectasis was made. On December 18, bronchoscopy was performed. The left main bronchus contained a tumor which almost filled the lumen. It was rough, vascular and priable. The appearance suggested malignancy.

The following microscopic report on the section was made: The tissue fragments showed a malignant neoplasm composed of small hyperchromatic cells, containing scanty cytoplasm and relatively large round nuclei which were either solid or vesicular. The outlines of the cells were poorly delineated though the predominating cell appeared round. The cells were disposed in small solid alveoli, in diffuse masses and in multiple rows about central cores of vascular fibrous tissue. The peripheral cells of the alveoli were often elongated with their long axes directed at right angles to the stroma. The latter was generally sparse. In places it was vascular, while in other places it was dense and hyaline.

A diagnosis of a malignant tumor, either carcinoma or endothelioma, was made.

December 22: Bronchoscopy was performed. As much as possible of the tumor was punched away piecemeal. The tumor bled freely.

December 23: The temperature decreased directly to 100 F.

December 28: X-ray examination showed complete atelectasis of the left lung. The heart and mediastinal contents were drawn to the affected side, with an elevation of the left side of the diaphragm.

December 29: Bronchoscopy was performed. The tumor appeared as before. Surgical diathermy, a bipolar electrocoagulating current was applied directly to

Fig. 3.—The condition of the lungs is beginning to clear up after bronchoscopy on Jan. 13, 1927.

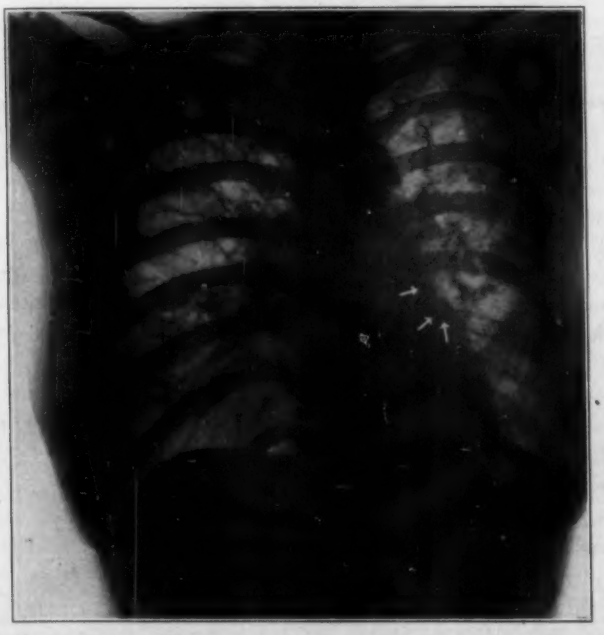

Fig. 4.—Showing the radium seeds in place on March 12, 1927. The condition of the lungs continued to clear up.

the tumor mass by means of a long electrode passed through the bronchoscope. Some of the tumor tissue was then removed with forceps. The temperature remained between 99 and 100 F.

December 3: The patient was doing very well. Considerable air was getting through the bronchus in the alveoli as indicated by physical signs and x-ray examination.

Jan. 7, 1927: The lung was much clearer. Bronchoscopy showed that the growth was much smaller. Diathermy was again applied.

January 13: X-ray examination showed that the lung was much clearer, especially at the apex. The heart was still drawn to the affected side.

January 18: The patient was allowed out of the hospital every day. The cough and expectoration had decreased markedly.

January 21: Bronchoscopy was performed. The growth was reduced in size. It was freely movable on a pedicle. It had lost its vascular character. The bronchoscope could be inserted beyond the tumor. The lower portion of the bronchus appeared normal. Iodized oil 40 per cent was injected, and x-ray examination showed evidence of numerous bronchiectatic cavities at the base. The temperature remained between 99 and 100 F.

January 25: The temperature was slightly elevated for one day. Bronchoscopy was performed. The growth appeared a little larger and more vascular. Diathermy was again applied.

February 1: The patient had a slight fever, but it was beginning to come down.

February 3: The patient was sent home, feeling well. The temperature was normal. There was no dyspnea, cyanosis, cough or expectoration.

February 24: Bronchoscopy showed that the growth was still present, but much smaller. It appeared to spring from the outer wall of the bronchus. Its longitudinal extent was about 1 inch (2.5 cm.). Diathermy was again applied.

March 12: Bronchoscopy was performed and three seeds of radium were inserted into the tumor mass through the bronchoscope. A total of 9.3 millicuries was used.

March 23: Bronchoscopy showed that the growth was a little smaller. One needle was removed. The other two could not be located.

May 11: Bronchoscopy was performed. The growth was smaller.

July 5: Bronchoscopy was performed. The growth had diminished in size. It was seen to be at the junction between the upper and main left bronchus. Diathermy was again applied.

November 7: Bronchoscopy showed that the growth was smaller than at any time, and much less vascular. There was a globular, seemingly pedunculated mass, and a sessile growth running along the lower circumference of the bronchus of the upper lobe.

Jan. 4, 1928: Bronchoscopy showed that the growth was larger. It was burned with diathermy.

January 30: Bronchoscopy was performed. The mass was markedly diminished in size and appeared entirely white. X-ray examination showed the lungs to be completely clear. Diathermy was again applied.

March 20: Bronchoscopy was performed. A small white area was seen at the site of the tumor. The appearance was that of an area of scar tissue. The patient's general condition was excellent at that time. She had gained 40 pounds (18.1 Kg.) since the relief from this last attack.

Fig. 5.—Showing two radium seeds still visible on Jan. 25, 1928. The lungs are clear.

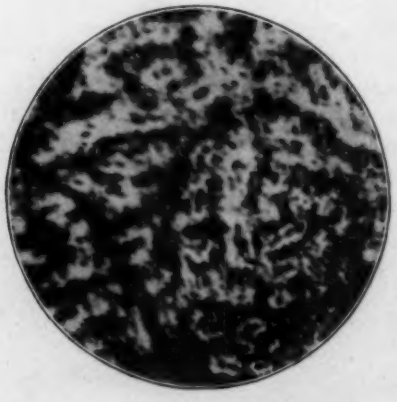

Fig. 6.—Section of the tumor.

COMMENT

This is, then, a case of carcinoma situated in the left main bronchus which caused attacks of complete atelectasis of the lung over a period of four years before treatment was instituted. The treatment consisted of frequent applications of surgical diathermy and the insertion of radium seeds directly through the bronchoscope. It is interesting to note that the morning after the first bronchoscopy some of the bronchial obstruction was relieved; the temperature came down to almost normal, and remained so. The atelectasis gradually cleared up, and with it, the dyspnea, cyanosis, cough and expectoration. The diathermy seemed to be more effective than the radium. It took fifteen months before it was thought the tumor had disappeared. This does not, however, mean that the patient is cured, but merely that, as yet, there is no local recurrence, and there are no evidences of metastasis in any other part of the body. The tumor, while malignant, was not a rapidly growing or metastasizing one.

It would appear that such bronchoscopic treatment is indicated in cases in which the tumor is limited for the most part to the bronchial mucous membrane and does not involve to any great extent the peribronchial tissue. In other words, such treatment would be guided by the size of the tumor. The position of the growth is another consideration. If the growth is very near the hilum, it would be a rather dangerous procedure to insert radium needles, which is done blindly to a certain extent. These might perforate the mediastinum or, because of the proximity of the mediastinum, affect the structures within it. If the growth is very far toward the periphery, it is possible that the bronchoscope would not reach it.

CONCLUSION

1. A case is here reported of complete atelectasis of the left lung due to blocking by a carcinoma of the left main bronchus.

2. The tumor was seemingly entirely removed by the use of radium seeds and the application of surgical diathermy through the bronchoscope.

3. The patient is symptomatically entirely relieved to date.

4. The case illustrates the importance of bronschoscopy in the diagnosis and treatment of patients who have tumors of the lung.

5. It also illustrates the importance of bronchoscopy in investigating the etiology of atelectasis of the lung.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. HEUER, SINGER, LILIENTHAL, MEYER AND KERNAN

DR. WILLIS S. LEMON, Rochester, Minn.: Since May, 1925, in the Section on Thoracic Medicine of the Mayo Clinic seventy-seven cases of primary pulmonary and bronchial malignant conditions have been observed. Eleven of these cases have been confirmed at necropsy, twenty-seven by biopsy on tissue removed through

the bronchoscope, one by tissue expectorated and preserved by the patient, and the remainder by examination of metastatic lesions, especially in lymph nodes, or by cytologic study of excretions from the bronchi or pleural spaces.

As a result of cooperative work, diagnosis has been made much more precise and accurate, treatment has been instituted more promptly, the interpretation of roentgenograms has been more accurate and more significant of the pathologic reaction producing opacities, and finally, it has been possible for the clinician to create a diagnostic picture characteristic of primary disease and to distinguish it from secondary or metastatic involvement. In the latter, symptoms are rare until late in the disease or unless the secondary growth is near the hilum when irritation produces paroxysmal nonproductive cough. It is rarely discovered by general examination unless the lesion is large or suppurative or in close approximation to the wall of the chest. Many metastatic nodes 2.5 cm. or more in diameter may lie within the lung and defy discovery except when seen in the roentgenogram. A primary malignant condition, on the other hand, is characterized by irritative nonproductive cough, becoming productive first of mucoid, then mucopurulent or purulent, and finally, of hemorrhagic sputum. The cough is disproportionate to objective symptoms but soon becomes associated with subjective and objective thoracic pain, dyspnea, fever, hoarseness and loss of weight. Clinical examination reveals the lesion with comparative accuracy; bronchostenosis was present in nineteen of twenty-seven cases proved by examination of tissue removed through the bronchoscope. In sixteen of the twenty-seven examinations squamous cell epithelioma was found; in ten, adenocarcinoma, and in one, lymphosarcoma, all of high grade malignancy.

Vinson and Moersch, who have done all of the bronchoscopic work, have been able to formulate a diagnostic picture fairly characteristic of the lesion, although they recognize that inflammatory and malignant lesions may be similar enough to be confusing until examined microscopically. Their reports to the clinicians and surgeons have been more significant than that on the results of any other examination. Every week at least one example of the value of bronchoscopy in clinical and surgical work is repeated.

Dr. Hermann Fischer, New York: I would like to report a case which has some points in common with the one that Dr. Willy Meyer just presented.

A. M., aged 39, was admitted to Lenox Hill Hospital on Oct. 11, 1927. At the time of admission, his chief complaint was pain in the left thigh, of about five days' duration. The pain was present constantly whether he lay down, walked or stood. He did not recall trauma of any kind. Otherwise, he was in good health. The family history was negative. The patient said that he always has been well except for an attack of grip four weeks ago. He had not had venereal infection. The only outstanding feature in his clinical history was loss of 26 pounds (11.8 Kg.) in weight, and also a pain in his left thigh.

A fairly well developed man, not appearing acutely ill was seen. The pupils were slightly unequal and reacted to light and accommodation very sluggishly. There was no glandular enlargement of the neck. The thorax was fairly well formed. It had a slight diminution of expansion of the upper right side of the chest. Over this region, both anteriorly and posteriorly, there was dulness with bronchial bleeding. Râles were not present. The rest of the chest was resonant and tactile fremitus was normal. The heart was not enlarged. The pulse was regular in rate and rhythm. The second aortic sound was accentuated. Tenderness, rigidity or masses were not felt in the abdomen. The liver was slightly enlarged. The spleen was not felt. There was marked tenderness over the left

sacral iliac region. The extremities were normal. The results of the rectal examination were negative. A provisional diagnosis was made of tuberculosis of the apex of the right and sacro-iliac strain.

The blood count showed: 4,000,000 red blood cells with 75 per cent hemoglobin; 7,200 leukocytes; 81 per cent polymorphonuclears; large lymphocytes, 11 per cent; small lymphocytes, 3 per cent; monocytes, 3 per cent; myelocytes, 2 per cent. Changes were not observed in the red cells. Chemical examination of the blood did not show any important changes. Urinalysis showed the urine to be clear with a specific gravity of 1,015, no albumin, sugar or casts and few white blood cells. The urine showed a little albumin and 0.2 per cent sugar and a few hyaline casts only once. The Wassermann reaction was negative. The sputum

Fig. 1.—Carcinoma of the upper lobe of the right lung taken October 14.

was mucopurulent, but several examinations for tubercle bacillus were negative. There was no evidence of neoplastic tissue.

On October 14, a stereoroentgenogram of the sacro-iliac region was negative. Examination of the chest showed an area of greatly increased density, occupying the upper third of the right lung field, with a sharply demarcated inferior margin and normal lung markings at the base. This area of increased density was equal to that of the heart, and was probably due to consolidation. Tumor, of course, was considered as a possibility.

On October 24, in the stereoscopic films of the chest, a slight decrease in density was noted in the involved area at the apex of the right lung. This is especially noticed in the films made of the dorsal spine. A lemon-sized area with sharply demarcated margins in the inner zone of the apical region was seen.

Careful examination of the stereoscopic views of the spine and chest did not show any evidence of metastatic involvement.

Bronchoscopy was performed by Dr. Kernan. The right bronchus was distorted by pressure. The bronchus to the upper lobe of the right lung was completely closed by outside pressure making it impossible to introduce a bronchoscope. This condition suggested a neoplasm.

A diagnosis of carcinoma of the upper lobe of the right lung was made. The patient was transferred to my surgical service.

As the patient's general condition was excellent and the only hope of saving his life was the removal of the growth, I decided to attempt it by a two-stage lobectomy. The operation was performed on Oct. 27, 1927. A semicircular flap

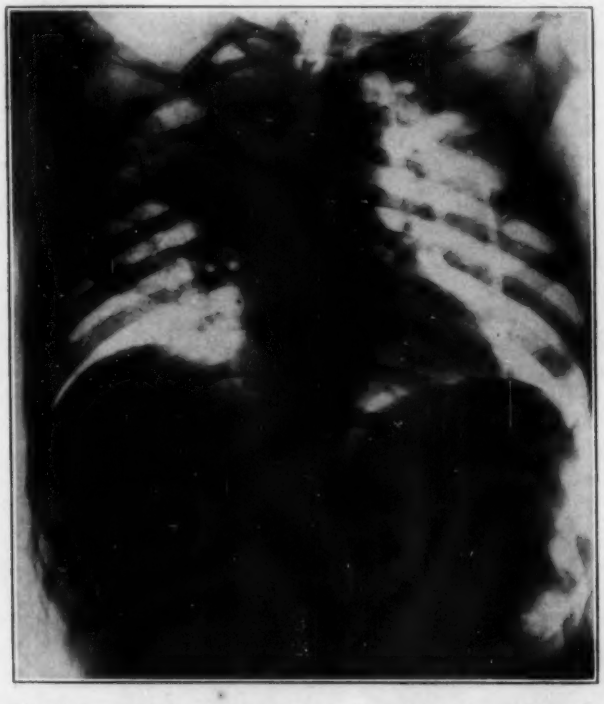

Fig. 2.—Carcinoma of the upper lobe of the right lung taken October 24.

incision was made on the anterior surface of the chest from the right of the sternum to the right axiliary line along the fifth intercostal space. The pectoralis major and minor muscles were separated from their attachments to the ribs, and the second, third and fourth ribs were resected. Under intrapharyngeal positive pressure, the right pleural cavity was opened. The upper lobe which was atelectatic contained a very hard mass compressing the eparterial bronchus. The lobe was adherent to the dome of the pleural cavity by numerous adhesions which carried blood vessels of some size which could be splendidly demonstrated by the Cameron light. The mediastinal tissues were not invaded by the growth. As the condition seemed to be operable, I proceeded to tie off and cut the adhesions; while doing this, the patient's condition, which was good up to this time, suddenly became alarming and I was compelled to stop. Before the wound was entirely closed, the

patient died. All attempts at rescussitation by direct massage of the heart and by injection of epinephrine into the heart failed.

The interesting feature of this case was the absolute lack of subjective signs in a tumor of the upper lobe of the lung, which had grown to considerable size. There was a complete absence of cough and sputum. The only subjective symptom which had induced the patient to seek admittance to the hospital was the inexplicable pain in his thigh and the loss of weight. Only a careful x-ray examination and a bronchoscopy cleared the picture and led to a diagnosis.

DR. HAROLD BRUNN, San Francisco: I would like to report two cases in relation to Dr. Heuer's paper, which are of some interest, especially from an x-ray diagnostic standpoint.

The first patient entered the University of California Hospital in 1925. He was a young man, aged 25, whose symptoms began three years previously with a dull aching pain in the right midback aggravated by stooping. Two months before he entered the hospital he developed numbness and tingling in the legs up to the thighs, and stiffness of the legs. Two weeks previously, he was unable to urinate. On examination, there was an area of dulness extending from the border of the right scapula; rigidity and pain on motion; the right lower leg was spastic, and dragged on walking. There was pain in the chest below the level of the fourth thoracic segment; touch was diminished and the lower reflexes were hyperactive. X-ray examination of the chest showed a smooth, round tumor the size of a small orange in the upper part of the chest; the lateral view showed it apparently springing from the vertebra. The patient was first operated on by Dr. Naffziger through a laminectomy incision. At this time a part of the growth was removed, and proved to be osteochondroma arising partly from the intervertebral disks. Later, the chest portion of the tumor was removed successfully with relief from pressure symptoms at the present time.

The second patient entered the University of California Hospital in 1927. A comparison of the x-ray pictures showed an exactly similar picture both as to location and appearance. He was 64 years of age and was suffering from amebiasis. X-ray examination of the chest was made because of his constant complaint from pain in the right shoulder and upper right side of the chest which required the use of narcotics constantly. The film showed a tumor in the upper part of the chest the size of an orange, in every way comparable both as to size, smoothness of outline and density with the preceding case. When the films were compared, one could hardly distinguish one from the other. A diagnosis was made again of osteochondroma, arising from the intervertebral disks. On operation, however, a cyst was found arising apparently from the mediastinum posteriorly, but its exact origin could not be determined. On microscopic examination, no definite pathologic process could be found. Its removal was difficult.

A. L. LOCKWOOD, Toronto: We had so little time this morning to discuss Dr. Harrington's paper. There was one point which I think Dr. Harrington might have stressed and which I had hoped he would this afternoon—that dealing with the phrenic nerve in lesions of the diaphragm. I think we should be particularly thankful and grateful to Dr. Harrington that he has brought to our minds another rare type of upper abdominal lesion which probably a great many of us have been overlooking. In January, 1918, it was pointed out to the Congress of Surgeons in Paris that after having dealt with about 165 abdominal thoracic lesions we had been so struck by our inability to save the patients, even with all resuscitory measures available, until almost by accident we discovered that it might be possible by injecting alcohol into the phrenic nerve to overcome the shock. This was done in five cases, with extraordinary benefit, and at that meeting it was

recommended that this procedure be adopted in all cases. I have recently had an opportunity to carry it out in one other case of injury to the diaphragm.

As regards the symposium on tumors, I think an extraordinary advance has been made in the knowledge of thoracic tumors. I would agree with the last speaker that it is probably in cases of benign tumors that the most startling results will be obtained.

Dr. Lemon, I am sure, will recall two or three cases we had together, particularly one of a man, an eminent ball player—whom you would all know by name—who came to the clinic with his wife who had rheumatism. The examiner noticed that this man kept spitting into the sink. He was going to speak to him about it when he happened to notice that there was a light reddish tinge to the sputum. He immediately turned his interest to the man and said, "How long have you been spitting up this sort of thing?" The man told him about three weeks. The examiner asked him if he had any cough, and the man replied, "Oh, a little bit, but I think it is due to smoking cigarets; I run a cigar store and smoke all day." As a result that man was immediately turned over to Dr. Lemon who gave him a medical examination, and an x-ray picture revealed an early primary carcinoma of the left lung. There was a little difference of opinion as to what should be done, and we decided to give him x-ray therapy. When he returned in nine weeks he had a much more extensive lesion. An operation was performed, but the growth was found to be too extensive; it was a widespread carcinoma. We saw this patient early; there was no loss of weight and the only symptom was a reddish-tinged sputum and a peculiar hacking cough.

Anothing interesting case is that of a prominent newspaper man on whom we performed an operation and found a teratoma in the right upper and anterior portion of the apex of the right lung. Microscopically, a section was identical with a teratoma which had been removed from the testicle five years before. It was almost impossible—I think Dr. Lemon will agree—to tell these two slides apart. The patient died six years after the operation on the testicle.

Four years ago I had a patient whose condition was discovered in a routine x-ray examination. We always have an x-ray picture made of the chest of all patients coming under our care. In this woman, we found a small tumor on the right side, low down anteriorly and medially. Thoracotomy was advised, and we found a tumor that I should think would measure from about 4 to 5 cm. in circumference on the anterior and medial aspect of the lung. It was easily dissected free, but had a pedicle about the size of my index finger running into the main bronchus. I did not dare to remove the tumor. That was four years ago. Each year since I have had an x-ray picture made of the woman's chest. Only in the last eight weeks has she developed fluid in the right side of the chest. I have seen the x-ray pictures, although I have not seen the patient. Her physician reported to me that her condition started with a cold; that he thought it was a pleural condition and that the fluid removed was not blood-stained.

There are two other cases I would like to mention here. We had a case of aberrant goiter that had been observed over a period of five years. Eventually, we determined that the patient was getting carcinoma in this growth within the lung, and a two-stage operation was undertaken. At the end of the second stage, the tumor, which measured probably 5 or 6 cm. in diameter, was freely mobile, attached only at one portion anteriorly. The growth was in the right lung on the anterior and medial border. This operation was performed before a group of surgeons and all were allowed to come to the table. It looked as if all we had to do was to shell it out. The woman was in good health generally. All at once we heard a sucking noise, the patient collapsed and was dead in an instant. We believed that death was due to an air embolism.

Since that I have had one other patient with a most extensive growth involving the right lung and wall of the chest high up. It was discovered after the man had had so-called neuritis for three years. It was too late to undertake an operation. He was nervous and irritable; he had a tremor and a high basal metabolic rate, and he had lost weight. At autopsy, we found carcinoma in an aberrant goiter.

DR. W. H. STEWART, New York: I do not know that I have ever attended a meeting of a society where there has been such harmonious opinion. However, I do not understand Dr. Singer's attitude toward the roentgenologists. We are evidently set aside without proper thought, for he ventures to direct us not only in the position in which he wants the patient placed, but he also instructs us regarding the character of the tube, whether it should be soft or hard ray. It seems to me that unless he is a roentgenologist himself, he is delving into problems which should be left to the men who specialize in such work and who are competent only after years of experience.

I believe that in all such problems as have been mentioned here the roentgenologist should be one of the consultants and should be as active in the clinical diagnosis as the bronchoscopist, surgeon or internist.

Dr. Singer's point of injecting air into the pleural cavity is an old trick and has been done for many years in an attempt to make a diagnosis of pleural carcinoma. It is an excellent method and it only illustrates my point that these patients should be studied carefully. Often they must be observed many times over a period of months before we can come to accurate conclusions. One cannot make a snap diagnosis. In many of these cases of globular shadows in the chest it is extremely difficult and unreasonable to expect to make a diagnosis of the character of the pathologic process on the basis of the x-ray examination alone. The result of the roentgen studies must be combined with all other observations before any definite conclusion can be made.

Following along these lines, I have a case in which this point is well illustrated. The patient had a tumor in the upper lobe of the right lung; there was some question of its being malignant. I watched this patient over a considerable period of time and finally obtained a roentgenogram showing two shadows in the opposite lung; this was sufficient to justify a diagnosis of a metastasis, proving the diagnosis of the original lesion. It is only by working constantly on these cases that we can attempt to make a correct diagnosis.

DR. LEO ELOESSER, San Francisco: I should like to mention briefly the case of a patient who presented a globular shadow such as Dr. Lilienthal showed and such as the second picture of Dr. LeWald showed. We had had him under observation for a period long enough to satisfy Dr. Stewart, namely, six years. Aspiration of the shadow yielded a chocolate colored fluid. Last year we decided to open the cyst. It was filled with chocolate colored fluid. The wall consisted of a thick, fibrous membrane. After repeated inoculations into guinea-pigs, we finally demonstrated tubercle bacilli. The cyst had remained at about the same size for six years.

DR. GEORGE J. HEUER, Cincinnati: With regard to Dr. Lockwood's question, I have not yet assembled all the cases of intrathoracic or aberrant thyroid, but I have gone far enough to know that malignant degeneration of these tumors has not been uncommon.

DR. J. J. SINGER, St. Louis: When we make a roentgen study, it has no reference to one, two or three divisions, whether we label ourselves roentgenologists or medical men or surgeons. I think we all realize that none of us are clever enough alone and that it is only by a combination of medical men, x-ray

men, bronchoscopists, laboratory men and others, that it has been possible to tackle this problem as we have today.

With regard to telling the roentgenologist what to do, to become a little more personal, we work with Dr. Sherwood Moore—I am sure you know him—and many of these problems have been experimental problems in which he takes the part only of interpreting the ex-ray picture. If he were to spend the time we spend, two or three hours on one or two cases, you can readily understand that it would be a physical impossibility to run his department. But we have learned a great deal from the radiologists, and what little I have shown on the plates I must say I have learned in part from them.

HODGKIN'S DISEASE OF THE NECK AND MEDIASTINUM

BILATERAL CERVICAL OPERATIONS; MEDIASTINOTOMY

WILLIAM LERCHE, M.D.
ST. PAUL

On Oct. 18, 1910, I examined a woman, aged 24, who complained of a lump which she had discovered two days previously above her left collar bone. She had measles, scarlatina and diphtheria in childhood and had been somewhat anemic between the ages of 16 and 18, but otherwise had been in good health. Her family history was without importance.

The results of the general examination were negative, and glandular enlargement was found only in the left supraclavicular nodes. My diagnosis was enlarged lymph nodes, probably tuberculous.

On October 20, I removed a mass of enlarged nodes from the supraclavicular space, through an incision above the clavicle. The pathologic report was: lymphoma, probably Hodgkin's disease (fig. 1). The patient was instructed to report for observation, but she did not appear until six months later, when the condition recurred in the same region. On April 17, 1911, an incision 15 cm. long was made through the old scar extending from several centimeters above the outer end of the clavicle, curving over the inner end of the latter to the midline of the sternum below the sternal notch. Through this incision, masses of lymph nodes in the supraclavicular space and nodes of the upper paratracheal chain were removed. The pathologic report was: lymphosarcoma? Hodgkin's disease? She had a slight recurrence in the left supraclavicular space three years and eight months later, and was operated on Dec. 8, 1914. This operation was followed by a series of x-ray treatments. A third recurrence took place about a year later, and on Jan. 3, 1916, I removed masses of nodes from the left supraclavicular space and paratracheal nodes, which again was followed by x-ray treatments. In November, 1916, the patient was married, and in 1917, gave birth to a healthy child. In 1919, she returned with a lump above the collar bone on the right side, and on Aug. 1, 1919, through an incision similar to the one employed on the left side, glandular masses were removed from the supraclavicular space and also nodes of the paratracheal chain. In 1921, she returned, complaining that for about a month she had had dyspnea, with a rattling noise in her chest while sleeping, hoarseness, occasional slight difficulty in swallowing, and itching. The dyspnea increased when she lay on the right side. Slight exertion brought on wheezing. There had been no headache, cyanosis or edema. The Wassermann reaction was negative. Roentgenograms revealed a large mass apparently jutting forth from the mediastinum and occupying a large part of the right side of the chest cavity (fig. 2). A diagnosis of mediastinal Hodgkin's disease was made. On May 11, 1921, under intratracheal insufflation anesthesia, a curved incision beginning in the lower part of the scar from the former operation in the right supraclavicular space, extending along the midline of the sternum and onto the fourth rib was made and the flap dissected. It was my intention to resect the second and third cartilages and ribs, but after cutting the cartilage of the second rib and freeing the rib, I found that by retracting the latter, sufficient space was gained to remove a large part of the mass. The pleura was accidentally torn, and by putting my hand through the opening I found the mass free except for its mediastinal attachment. The patient

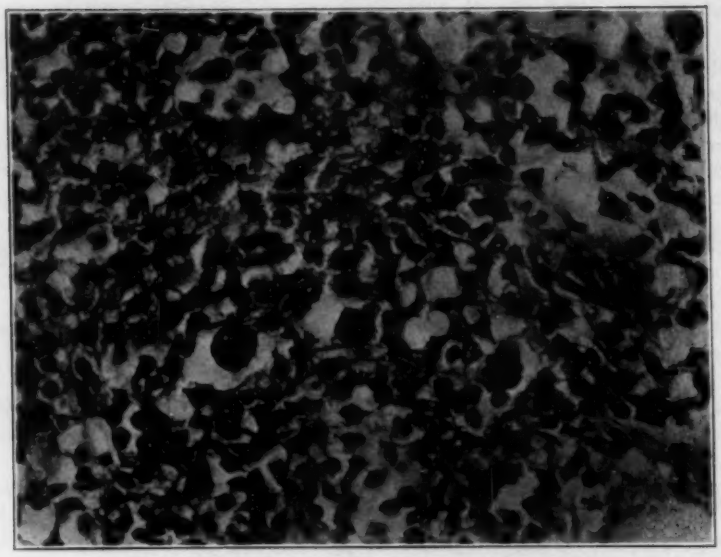

Fig. 1.—Section of cervical lymph node, 1910.

Fig. 2.—Hodgkin's disease involving the right tracheobronchial nodes; the paratracheal nodes are not involved.

made an uninterrupted recovery and was completely relieved of her dyspnea, hoarseness and dysphagia immediately after the operation. The itching disappeared within a week. The pathologic report was: Hodgkin's disease. Beginning May 27, 1921, she received a series of x-ray treatments, and another series beginning March 14, 1922. Further treatment has not been given. Examination of the patient once a year has not revealed any new development, and the shadow of fibrous tissue seen in a recent roentgenogram (fig. 3) has remained unchanged since the treatment in 1922. There has not been any enlargement of the spleen, liver or of any lymph nodes except those already mentioned during the years the patient has been under my observation. She has never complained of any throat trouble, and she still has her tonsils intact. The cosmetic result from the operation was good, but there was a relatively slight x-ray burn. The patient is in excellent health.

Fig. 3.—Roentgenogram taken in February, 1928, showing that there has not been any change in the shadow since 1922.

The pathologic material removed at the operations in 1910, 1914, 1919 and 1921 (the material from 1911 and 1916 is missing), has been examined by Dr. E. T. Bell and Dr. J. S. MacCarthney of the University of Minnesota.

The roentgenogram of the chest (fig. 2) does not show any recurrence in the right paratracheal nodes while a dense shadow will be noted at a level corresponding to the site of the right tracheobronchial nodes (insert, fig. 4). The mass apparently sprang from the latter nodes, and, pushing the plural cover of the tracheobronchial space ahead of it, projected laterally into the chest cavity on the right side, where it grew in all directions except mesially. At the time of operation, I found

the mass in close contact with the ribs anteriorly and the sternum, and although a large portion of the mass was left behind, there was complete relief from all symptoms immediately after the operation. The back pressure, particularly as it was exerted on the right bronchus and probably the trachea by the growing mass crowding the anterior wall of the chest, was therefore responsible for the symptoms. The hoarse-

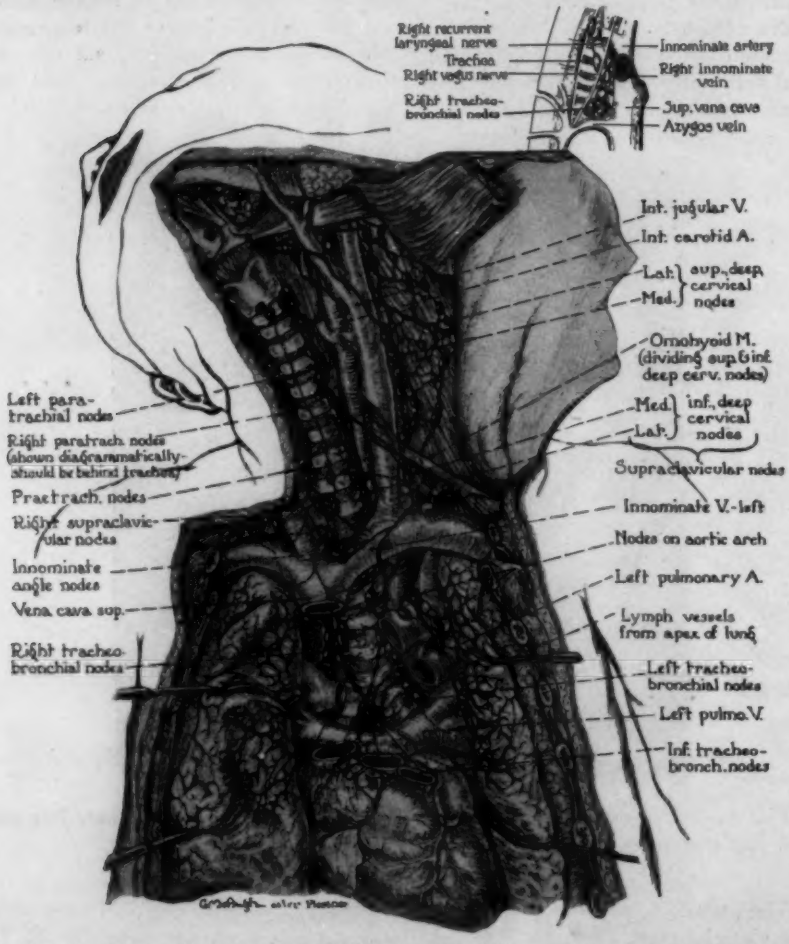

Fig. 4.—Some of the nodes and lymphatics of the neck and mediastinum, with particular reference to the supraclavicular nodes and the areas from which they may receive lymphatics. (Modified after Most, with additions from Bartels, Sukiennikow, Beitzke and with some observations of the author.)

ness and the occasional dysphagia was due to pressure on the right recurrent laryngeal and the vagus nerves (see insert, fig. 4).

Lack of etiology prevents definite classification of Hodgkin's disease. The exciting agent is thought to enter through the mucous membranes

or through lesions of the skin. In the majority of cases the mucosa of the mouth, nose and throat is the portal of entry, the clue to the latter being the site of the regional nodes first obviously enlarged. The cervical nodes were the first to show enlargement in from 50 per cent (in 120 of Ziegler's cases [1]) to 86.1 per cent (in 173 of Burnham's cases [2]). The axillary, abdominal, mediastinal, inguinal and other nodes are less often primarily affected.

The supraclavicular nodes (the inferior deep cervical, fig. 4) were the first to show enlargement. These nodes and the various sources from which they may receive tributaries are briefly described. The nodes, divided into a medial and laterial group, are situated in the triangular space bounded by the posterior belly of the omohyoid muscle above, the clavicle below and mesially by the internal jugular vein. They receive the efferent vessels from the lateral group of the superior deep cervical nodes, some from the axillary group, and according to Küttner,[3] occasionally from the tongue. (There is no flow of lymph from the supraclavicular to the axillary nodes.) Lymphatics from the upper surface of the liver, the anterior part of the diaphragm and diaphragmatic pleura, according to Kuttner,[4] communicate with the supraclavicular nodes (particularly with those on the left side) through the internal mammary lymph vessels and nodes, which explains the metastasis from carcinoma in the upper part of the abdomen, which may occur in the left supraclavicular nodes. In a case of carcinoma of the lower part of the right lung, I found metastasis in a left supraclavicular node, which may have occurred by the route just mentioned or through the inferior tracheobronchial nodes, which drain the lower and middle part of the lungs and anastomose freely with the lateral tracheobronchial groups. Lymphatics from the latter nodes communicate with the supraclavicular group. Of particular interest in this case are the tributaries from the tracheal mucosa through the pretracheal and paratracheal nodes. The pretracheal nodes, placed on the anterior surface of the trachea below the thyroid isthmus, receive lymphatics from the anterior part of the tracheal mucosa, the lower part of the thyroid gland and prelaryngeal nodes, their efferent vessels draining either directly into the supraclavicular group or into the paratracheal chain. The paratracheal nodes (recurrent chain) are situated in the groove between the trachea and the esophagus and connect below with the tracheobronchial groups. They drain the larger part of the tracheal

1. Ziegler, K.: Die Hodgkinsche Krankheit, Jena, 1911.

2. Burnham, F.: Hodgkin's Disease, J. A. M. A. **78**:1445 (Oct. 30) 1926.

3. Küttner, H.: Ueber die Lymphgefäsze und Lymphdrüsen der Zunge, Beitr. z. klin. Chir. **21**:732, 1898.

4. Küttner, H.: Die perforirenden Lymphgefässe des Zwerchfells und ihre pathologische Bedeutung, Beitr. z. klin. Chir. **40**:136, 1903.

mucosa and part of the esophagus, their efferent vessels from above coursing downward and outward to a level corresponding to the supraclavicular space to enter the supraclavicular nodes, and those from below this level, coursing upward and outward to drain into the latter nodes. Some efferent vessels from the pretracheal and paratracheal nodes drain into the group of nodes at the innominate angle. The lymphatics from the lower part of the trachea and its bifurcation drain into the tracheobronchial groups, and efferent vessels from these groups ascend and may drain into the supraclavicular nodes, either directly or through paratracheal nodes, or by way of nodes at the innominate angle. Trunks from the innominate group run along the upper border of the

Fig. 5.—Examples of variations from the normal course of lymphatics as described in the text. (Most.)

innominate vein to the left and may anastomose through nodes with lymph vessels from the left side and thence proceed to the venous angle (angulus venosus), i.e., the angle at the junction of the subclavian and the internal jugular veins. Most [5] found lymph vessels from the apex of the left lung that did not enter the nodes at the left tracheobronchial angle, but passed into nodes laterally on the aortic arch and ascending to the venous angle emptied into the vein directly or through a supraclavicular node. The uppermost node in the right tracheobronchial space communicates with nodes at the innominate angle (Bartels [6]).

5. Most, A.: Untersuchungen über die Lymphbahnen an der oberen Thoraxapertur und am Brustkorb, Arch. f. Anat. u. Entwickelungsgeschich., 1908, vol. 1.

6. Bartels, P.: Das Lymphgefässsystem, Jena, 1909.

The lymphatics are subject to many variations. On injecting the upper part of the tracheal mucosa, Most [5] not infrequently found lymph vessels coursing downward on the outer wall of the trachea to enter a paratracheal node at a considerable distance below. In one instance such a lymph vessel passed downward to empty into one of the tracheobronchial nodes. Occasionally, he found lymph vessels crossing to the opposite side (fig. 5). Although variations such as those mentioned may occur, Beitzke,[7] Most and others have shown by experiments on animals and injections into cadavers, that there is no flow of lymph from the cervical lymphatic system into that of the lungs and bronchi as was formerly thought. Thus, Grober [8] injected 10 cc. of an emulsion of india ink into the tonsillar region in dogs, and at necropsy found the lungs dotted black and the corresponding nodes containing particles of india ink. He concluded that the injected material had been carried to the lungs and thoracic nodes by the lymph current from the area of injection. Beitzke, in similar experiments, showed that there was no communication between the cervical lymphatics and those of the lungs and bronchi, and that in the animals in which particles of india ink were found in the lungs and corresponding nodes, this was due to the fact that part of the fluid used for injection had been aspirated, carried through the bronchial mucosa and thence by the lymphatics into the lungs and nodes. All lymphatics from the head and neck as well as those from the body, converge toward the base of the neck, collect into trunks and empty themselves into the venous system at the angle of the subclavian and the internal jugular veins on both sides. The original portal of entry in this case was probably the upper part of the tracheal mucosa.

As the disease remained relatively localized throughout, it would be of interest to follow the course of its spread in the lymphatic system. The provocative agent originally journeyed with the lymph current from the tracheal mucosa through the paratracheal to the supraclavicular nodes; later its advance was downward i.e., against the lymph current to the right tracheobronchial nodes. That the provocative agent may have escaped through an efferent trunk from the supraclavicular nodes into the venous circulation and through the right side of the heart and pulmonary artery to the lung, there emerging from the blood vessels into tissue spaces and carried by the lymphatics of the lung to the tracheobronchial nodes, is presumably possible. However, as only the right side was apparently involved, spread by this route seems less likely. An extension of surface infection to the lower part of the

7. Beitzke, H.: Ueber den Weg der Tuberkelbazillen von der Mund und Rachenhöhle zu den Lungen, mit besonderer Berücksichtigung der Verhältnisse beim Kinde, Virchows Arch. f. path. Anat. **1**:184, 1906.

8. Grober, J. A.: Die Infektionswege der Pleura, Deutsches Arch. f. klin. Med. **68**:296, 1900.

tracheal mucosa, which is drained by the tracheobronchial nodes, or by lymph carried downward by aberrant vessels already described, or a retrograde spread, are possibilities to be considered. Foreign matter in the lymph is not transported against the current. When therefore, in an area where obstruction to the normal flow of lymph exists and the spread of the disease is retrograde, it is really an extension by contiguity along short connecting lymphatics and between nodes in close proximity. According to Most,[9] such retrograde spread rarely takes place in tuberculosis of the nodes. The absence of recurrence in the right paratracheal chain would seem to preclude the probability of extension by contiguity to the tracheobronchial group unless it had taken place prior to their removal. As recurrence took place within six months after the operation in 1910, and three years and eight months elapsed without recurrence ofter the paratracheal and supraclavicular nodes were removed in 1911, the question of regeneration of the nodes may be considered. There is decided difference of opinion in regard to the regeneration of the lymph nodes. Baum,[10] in recent experimental work on dogs, did not find any regeneration of nodes after extirpation, but that a network of lymph vessels may become established in from ten to twelve days. Most,[11] discussing the literature on this question, his own anatomic research work on the lymphatic system and clinical experience with excision of nodes in cases of glandular tuberculosis as well as in carcinoma, said that under certain pathologic conditions, there unquestionably may be an increase in the number of nodes and that after extirpation new nodes may form, but that this does not necessarily always take place. Some authors claimed that a small amount of fat left after the extirpation of pathologic nodes is sufficient for the regeneration of nodes and lymph vessels in that area. Most said that this has not yet been proved, and he thought it an overestimation of the regenerative ability of the lymphatic system.

Surgical treatment for Hodgkin's disease is not in vogue today, yet Yates,[12] an ardent advocate of early complete excision of the superficial nodes followed by x-ray therapy, has had good results and expressed the belief that recovery may be expected in 20 per cent of all cases.

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9. Most, A.: Die Topographie des Lymphgefässapparates des menschlichen Körpers und ihre Beziehungen zu den Infektionswegen der Tuberculose, Bibl. Medica., pt. C, 1908, p. 21.

10. Baum, H.: Folgen der Extirpation normaler Lymphknoten für den Lymphapparat und die Gewebe der Operationsstelle, Deutsche Ztschr. f. Chir. **195**:241, 1926.

11. Most, A.: Chirurgie der Lymphgefässe und der Lymphdrüsen, Neue Deutsche Chir., 1917, vol. 24.

12. Yates, J. L., and Bunting, C. H.: Results of Treatment in Hodgkin's Disease, J. A. M. A. **68**:747 (March 10) 1917.

The roentgen-ray therapy available and as applied in 1914 and 1916, did not check the progress of the disease in this case. The form and amount of irradiation used after the mediastinotomy in 1921, however, was sufficient to prevent recurrence of the disease up to the present time.

ABSTRACT OF DISCUSSION

DR. MCCARTNEY: This particular case has been of considerable interest to Dr. Bell and myself at the University of Minnesota, because, as you all know, the time of the natural course of Hodgkin's disease has been far exceeded in this case, the average course being only two to three years.

I have examined a number of lymph nodes, from this patient from each of the operations for 1910, 1916, 1919 and 1921, having three or four or five nodes or sections of three or four or five nodes from each operation. You can take those sections and mix them up and it is nearly impossible to tell from what particular year they came. They all show a very typical Hodgkin's disease. Some people might call it a lymphosarcoma, but what some people would call a lymphosarcoma we would call a cellular Hodgkin's disease. There is nothing to suggest tuberculosis in any node.

Now regardless of what your individual ideas may be as to what Hodgkin's disease actually is, whether you believe that it is a true tumor, a definite granuloma, an atypical form of tuberculosis, or what, does not seem to make any difference. The important thing in this case is that here is a patient who has had the disease for eighteen years and has been practically symptomless for the last seven years. My personal feeling is that Hodgkin's disease is a tumor, and that there is also an intimate relationship between the conditions called Hodgkin's disease, lymphosarcoma, endothelioma and leukemia. That is well illustrated in a recent case in our department in which we found these four different conditions in a single lymph node; there were perfectly typical leukemic and Hodgkin's infiltrations of the liver, also the type of bone marrow which is ordinarily associated with a myeloid type of leukemia. We have been much interested in following this particular case over this period of years.

DR. J. L. YATES: As suggested by Dr. McCartney, the name attached to any of the manifestations of malignant granulomatosis is of little moment because this protean disease has an almost indefinite number of mutations. The patient reported by Dr. Lerche, however, presents three noteworthy peculiarities: 1. Women of the age of this patient seldom develop the anatomic alterations peculiar to the form called Hodgkin's disease. 2. Patients, either male or female, are unlikely to retain the same type of lesion for so many years. 3. Exceptionally few patients survive so many operations and exposures to x-rays, and this one is reported as apparently well after such experiences. Her recovery would seem to approach the miraculous unless it can be explained. We, too, have had comparable experiences and sought a rational explanation so that it might be possible similarly to assist others to recover.

Virtually every one is subjected to the actions of the virus or viruses that provoke malignant granulomatosis. The majority are insusceptible. They suppress the virus without manifesting signs or symptoms of an infection. Three degrees of susceptibility occur among the minority. Those of least susceptibility develop spontaneously sufficient defense to suppress the virus, but only after exhibiting lesser signs and symptoms. They constitute the large proportion and achieve recoveries with, without, or in spite of, treatment. Those of moderate susceptibility develop pathognomonic manifestations. If they receive prompt and suitable treatment, all are enabled to live longer and more active lives, and some are assisted

to recover. Occasionally, the recoveries are dramatic and suggest the miraculous, but they are merely examples of unusual defensive powers developed under stress. Those of immoderate susceptibility constitute the smallest proportion. They are so lacking in defensive potentialities that their diseases appear in the more acute forms and, however treated, usually progress rapidly to death, though by exception are converted into more chronic forms.

Thus we have been forced to acquire much humility and to realize that therapy is impotent if defense is initially weak or has been permanently impaired. Greatest benefits are conferred on patients if the competence of their defense be estimated, and if the nature of the treatment be thereby determined.

Extirpations are, as a rule, as beneficial as they are radical and sometimes as they are multiple. Radiation is useful in palliation, particularly in reducing rates of metabolism, which are often high, in reducing temporarily the size of lymphomas, and rarely in relieving pruritus. It is injurious to lymph gland structures, which provide the large part of defense, and the more often patients are exposed to radiation, the greater the harm.

From studies extending over twenty-five years, we believe that patients as seriously affected as this one have recovered in spite of radiation and not because of it, and would have been better served with an antituberculosis regimen, including particularly well controlled exposures to sunshine.

DR. LE WALD: I would like to mention the work that Dr. L'Esperance at Cornell is doing in regard to the theory that avian tuberculosis is the cause of Hodgkin's disease. Dr. L'Esperance used some fresh material from a case of Hodgkin's disease which was inoculated into chickens and produced a typical histologic picture of Hodgkin's disease, and clinically gave the same appearance as that condition. On discussing this with Dr. James Ewing, he said that while this is still not entirely proved in a satisfactory way, it promises to throw some light on the etiology of Hodgkin's disease.

DR. BRUNN: I merely want to relate what might be a coincidence in regard to two cases of Hodgkin's disease in a man and his wife, both in the sixth decade. The woman was first seen with enlarged glands in the neck and abdomen, from which she died. Within the same year, her husband developed enlarged glands on the right side of the neck; the glands were removed en masse before they had grown very large and then roentgen treatment was given. These cases were reported also to be Hodgkin's disease. Roentgen treatments were continued after operation and there is no evidence of recurrence to date. Both of these cases occurred in 1918. Microscopic examination of both glands was made by Dr. Rusk, pathologist at the University of California Hospital.

DR. LERCHE: Recurrence followed in six months after the first operation, in which only supraclavicular nodes were removed. After the second operation, in which supraclavicular and paratracheal nodes were removed, there was no recurrence for three years and eight months. In the third operation, only supraclavicular nodes were removed, and following this operation, x-ray treatment was given. Recurrence took place in one year. In the fourth operation, in 1916, supraclavicular as well as paratracheal nodes were removed and followed by the same type of x-ray therapy. There has been no recurrence on the left side since, and I am inclined to ascribe the good result to the surgical intervention rather than to the x-ray treatment, as the latter was applied at that time.

THE RELATIONSHIP OF THE HEART AND LUNGS IN DISEASE

PAUL D. WHITE, M.D.
BOSTON

The relationship of the heart and lungs in disease is an important and interesting, though often neglected, study. These vital organs are more intimately associated than most other structures in the body, for not only are they interdependent functionally, but they are actually in contact anatomically, only the delicate tissue of the pleura and pericardium separating them. Thus not only will derangement of function of one organ affect the other if of sufficient magnitude, but disease or disturbance of structure of the one is apt to spread by contact to the other, or at least to displace it. It is to be remembered that the heart is, strictly speaking, a mediastinal organ along with its great vessels while the lungs are the structures that flank it on both sides, filling the chest proper. Associated with the fact that the heart lies more to the left than to the right of the midline is the division of the left lung into two lobes while the right lung has three. Other anatomic points of importance are the location of the heart mainly in the inferior half of the mediastinum with the pericardium and inferior vena cava, while the aortic arch with its branches, the superior vena cava, the trachea, the thymus gland and the upper part of the esophagus lie in the superior mediastinum. The pulmonary artery and its branches, the main bronchi and the bronchial lymph glands extend out from the mid-mediastinum. The thoracic aorta and esophagus penetrate the diaphragm posteriorly, while the main azygos vein is a right-sided structure before it enters the superior vena cava.

Finally, the most important reason for the consideration of the effects of diseases of the heart and lungs on each other is the necessity of recognizing the primary cause of chest symptoms and signs, which is not always an easy matter.

THE HEART IN DISEASES OF THE LUNGS

First I shall discuss the effect of various diseases of the lungs on the heart, beginning with acute conditions and finally taking up chronic illness and pleural involvement.

Lobar Pneumonia.—Lobar pneumonia is not only a disease of the lungs, but also a general systemic infection, and so exerts a twofold influence on the heart, the toxemia and septicemia being of greater importance than the local changes in this respect. The reaction of the heart depends on two factors: (1) the degree of the toxemia and (2)

the condition of the heart itself. With the heart in good or fair condition, its response to the demands of the illness will be good provided the toxemia is not overwhelming. The body itself is obliged to combat this toxemia, with the aid, in rare cases, of an antiserum injected into the blood stream. The heart does the best it can, and the toxemia that depresses it is not controlled by any amount of stimulation. Whether or not help can be obtained in critical cases by the use of digitalis is, however, still a moot question. Certainly it seems unwise to use the drug as a routine in every case. If the need should arise, it can be supplied quickly enough. Several points should be considered in discussing the use of digitalis in pneumonia. 1. Any drug or method of treatment given in pneumonia is apt to be awarded undue credit or blame because of the rapid change for better or worse that occurs usually spontaneously at the time of the crisis. 2. Digitalis is a poison itself, and to add poison of uncertain benefit to a system already full of toxin is not sound therapy. This drug alone will produce depression, malaise, anorexia and nausea, and may disturb cardiac rhythm. 3. When digitalis is used in pneumonia, it is generally given in such small dosage that it is probably without therapeutic effect. For example, two or three ampules of a digitalis preparation are given in extremis, intravenously or subcutaneously, or from 10 to 15 drops of the tincture of the preparation are given two or three times a day for a day or for two or three days. The knowledge that such doses will not save the lives of those with true heart failure who really need the drug, while larger doses will do so, makes it seem probable that these small amounts are but thrown away. Full and fairly rapid digitalization should be carried out in pneumonia if one decides to use the drug. Finally, in pneumonia as in other infectious diseases, and in accidents and surgical emergencies, it is not heart failure primarily that kills, but vasomotor shock, and digitalis is ineffective in combating vasomotor shock. Exceptionally there are persons, generally elderly, who have cardiac weakness or failure, to start with, because of heart disease. In these patients, digitalis therapy may be essential in the presence of auricular fibrillation; but even in such patients there is less beneficial influence from the drug in pneumonia than in the nonfebrile state, the ventricular rate in auricular fibrillation, for example, generally remaining high in spite of the use of digitalis. In other cases, also exceptional, the onset of auricular fibrillation during pneumonia may demand medication with digitalis; in these cases, however, quinidine sulphate may be preferable. The question of the use of digitalis in pneumonia is not settled; more light is needed.

Besides the toxic effect of lobar pneumonia on the heart, there is, in rare instances, an actual infection of the heart itself by the pneumococcus, taking the form of an acute bacterial endocarditis. This is almost

invariably a fatal complication, but rare cases have been recorded of aortic regurgitation developing during pneumonia, with survival. Pneumococcus pericarditis, purulent and usually fatal, is another rare complication of pneumonia.

Finally, the actual mechanical obstruction to the flow of blood in the lungs as the result of the lobar consolidation may affect the heart in two ways. There is an additional burden on the right ventricle to overcome the pulmonary hypertension; the larger the area involved, the larger is this load. When the heart is already exhausted by the toxic effect of the infection and by the anoxemic blood supply, this extra strain may conceivably favor a fatal outcome with massive involvement of the lung. The other effect of the pulmonary consolidation is the production of anoxemia due to lack of sufficient blood surface exposed to the oxygen of the inspired air, especially in relation to the raised metabolism resulting from the infection. This anoxemic blood flowing through the coronary arteries fails properly to support the overworked myocardium. However, it seems probable that neither the obstruction to blood flow through the lungs nor the rare septicemic cardiac or pericardial involvement are of as much importance in lobar pneumonia as is the toxic condition of the blood. The pleural complications of pneumonia as they affect the heart will be considered later.

Bronchopneumonia.—About the same thing may be said about the effect of bronchopneumonia on the heart as about that of lobar pneumonia. However, there is much greater variation due to the different grades of severity of the bronchial type; generally there is less strain on the heart, but in old people with coronary sclerosis in whom the disease is frequent the burden may be severe. The many infants who die of bronchopneumonia doubtless do so more as the result of the toxic effect of the infection than because of cardiac weakness. The use of digitalis in bronchopneumonia should probably be limited to aged people with definite cardiac weakness and to rare persons with special needs, as, for example, in persons with mitral stenosis and auricular fibrillation. As cardiac stimulants, all other drugs are inferior to digitalis except epinephrine which has a powerful but transient effect, and strophanthin or ouabain, which is also powerful but rather uncertain in action.

Acute Bronchitis.—Acute bronchitis has no significant effect on the heart. In a person with heart disease on the verge of failure, it may, like any other acute illness, precipitate trouble which may be corrected or prevented by the use of digitalis.

Whooping Cough.—Whooping cough may be mentioned for a moment here because it sometimes has been accused of having caused endocarditis or other heart disease. There is no evidence that heart disease has ever been produced by pertussis, although it is well recognized

that there is a considerable temporary strain on the right ventricle during the paroxysms of cough. The heart weathers the strain as a rule even when diseased, as with congenital defects or rheumatic lesions, and no enlargement results—only the transient dilatation at the time of the whoop. The rare circulatory accidents that do occur are vascular ruptures in brain or elsewhere.

Chronic Bronchitis.—Chronic bronchitis may or may not be a strain on the heart, depending on the severity and duration of the infection and on the age of the victim. The increase in pulmonary arterial pressure that results after years of chronic bronchitis causes right ventricular hypertrophy of varying extent, rarely so marked as in mitral stenosis. In older people with the development of emphysema, this strain on the right ventricle may prove too great and dilatation and failure follow with right auricular as well as ventricular dilatation. It is said that chronic pulmonary disease or chronic pulmonary arterial hypertension may lead to sclerosis and dilatation of the pulmonary artery. That is at least not always true as evidenced by some cases of well marked mitral stenosis with smooth pulmonary arteries. Without doubt, some of the prominence in the region of the pulmonary artery that is found by x-ray examination in such cases is not due to the shadow of the pulmonary artery, but to that of the infundibulum of the enlarged right ventricle.

Bronchiectasis.—Bronchiectasis has the same effect on the heart as have the extreme degrees of chronic bronchitis—the results of the strain on the right ventricle. The cyanosis that occurs so frequently in these conditions is only in part the result of cardiac failure. Undoubtedly a considerable and perhaps the larger amount is due to shunting of venous blood from the pulmonary artery to the pulmonary veins through lung tissue that is closed to contact with inspired air, or through dilated vessels in the center of which the blood cannot take in oxygen. The correction of cardiac congestive failure which is present by digitalis and rest will not dispel all the cyanosis or the clubbing of the fingers, for both of these conditions are due primarily to the right to left shunt of venous blood.

Emphysema.—Emphysema, so-called, whatever its primary cause, is also associated in chronic cases with heart disease or failure resulting from the strain on the right ventricle. Its frequent association with chronic bronchitis and with old age adds to the burden. As it is often difficult to determine how much of the symptomatology and how many of the signs are the result of pulmonary disease and how much the result of the heart strain and failure, it is rational to carry out a therapeutic test with digitalis, especially in older people. There is a definite entity known as "the emphysema heart."

Bronchial Asthma.—Bronchial asthma, if not severe or chronic, has little effect on the heart, but with long duration and in severe grade it undoubtedly produces a strain on the right ventricle comparable to that of bronchiectasis. The acute paroxysms may resemble somewhat the mechanism of whooping cough. Finally, emphysema results with the effects already discussed.

Pulmonary Abscess.—Pulmonary abscess generally has little or no effect on the healthy heart. Symptoms result from the toxemia and perhaps pleural involvement, and in rare cases from the endocarditis due to septicemia.

Pulmonary Infarction.—Pulmonary infarction varies in its influence on the heart, depending on its size. A large part of the lung tissue suddenly blocked off by embolism may cause death at once, probably as much the result of vasomotor shock as to the sudden strain on the right ventricle which must come with the rise in pulmonary arterial pressure. Experimentally, in anesthetized animals, much more pulmonary obstruction may occur with survival than in man. The element of nervous shock doubtless accounts for the difference. However, even in the experimental animal, pulmonary infarction may cause failure of the right side of the heart. With gradual infarction from thrombosis, the strain is probably much less for the same amount of tissue blocked off.

Massive Collapse.—Massive collapse of a lung or portion of a lung, if not associated with pleural fluid or air, will cause a displacement of the heart to the affected side without much circulatory embarrassment if the heart is normal.

Tuberculosis, Syphilis and Malignancy of the Lungs.—Three more types of chronic pulmonary disease are to be considered, but they can be passed over briefly. Tuberculosis of the lungs does not affect the heart except in very advanced disease when so much of the lung tissue is involved that intrapulmonary arterial pressure is raised and the right ventricle overburdened. There are two conditions of the heart often wrongly ascribed to the effect of tuberculosis of the lungs: (1) effort syndrome or nervous heart symptoms and (2) a small "drop" or "vertical heart." Any infection long drawn out in a nervous, often delicate person may produce effort syndrome due to the strain on a sensitive nervous system and not to any direct strain on the heart. Since it is often in slight ptotic persons that tuberculosis develops, a vertical or small heart, or so-called cardioptosis, is frequently discovered by x-ray or physical examination. The tuberculosis does not cause this, although the infection may exaggerate the weakness so often associated with such a condition. Many patients with phthisis show hearts normal in shape, position and size and many small or ptotic hearts are found in people

without tuberculosis. Inactivity of body doubtless does favor, however, muscular atrophy, and in this process the heart may be involved.

Syphilis of the lungs is rare and probably does not produce any effect on the heart. Ayerza has, however, described a condition consisting of sclerosis and dilatation of the pulmonary arteries, said to be due generally to syphilis. Failure of the right side of the heart and marked cyanosis resulting in the so-called "black cardiacs" has been described in this condition. In the rare cases of marked cyanosis with chronic pulmonary disease and emphysema that I have seen, syphilis apparently was not a factor, but in parts of the world, for example, South America, where syphilis is more common, Ayerza's disease may not be so rare.

Malignant disease of the lungs is very rare, and, except with massive involvement, does not influence the heart.

Diseases of the Pleura.—Diseases of the pleura affect the heart only as they may cause displacement by pressure or by retraction, or spread of infection by contact with the pericardium. This applies to acute and chronic pleuritis, with or without effusion, empyema or pneumothorax. Hydrothorax, serofibrinous effusion, pus or air may displace the heart to either side if in sufficient amount. As a rule the heart is not tilted much in the process, that is, the anatomic angle is not changed as it is in respiratory movements of the diaphragm. The heart is rather pushed as a whole, the mediastinum being easily displaced and the base of the heart not much more fixed than its apex. If the heart is much disturbed or twisted, the vessels may be kinked or compressed and thus the heart action be disturbed. This fact added to the loss of function of one lung and the encroachment of the heart volume on the other makes paracentesis to remove serum, pus or air at times an emergency measure. In chronic disease of the lungs and pleura, contraction of the tissue may pull the heart to either side or may even displace only certain parts of it. Roentgenograms, in demonstrating such conditions, are invaluable as they are also when, because of fluid, air or emphysema, it is difficult or impossible to ascertain the exact position of the heart.

Deformities of the Chest.—Deformities of the chest, as by scoliosis or by a funnel-shaped sternum, may displace the heart and embarrass its action, at times causing actual increase in size and favoring eventual failure. Very rare deformities or displacements of the diaphragm, as with eventration or hernia, may alter the position of the heart.

Care of the Heart in Thoracic Surgery.—In thoracic surgery, the heart is to be considered mainly as in general surgery. Preparation by rest and digitalization is indicated if there is definite or suspected cardiac weakness or auricular fibrillation, and not as a routine measure or for valvular or other heart disease without weakness. Local or general anesthesia, when skilfully administered, is well borne by the heart, even

when the patient is considerably diseased. Syphilitic aortitis and coronary thrombosis afford the chief risks. Asphyxia is the chief condition to avoid in anesthesia if the heart is weak. Ethylene as a general anesthetic seems to be particularly suitable for the heart and circulation. In cardiovascular emergency during or after operations, therapy directed to relieve vasomotor shock is generally much more indicated than is cardiac stimulation. Much digitalis has been wasted in such cases. Paroxysmal disturbances of cardiac rhythm during or after operations are generally transient and unimportant, though alarming to the surgeon—vagal pressure at times stops paroxysmal tachycardia, but as a rule the disturbances cease spontaneously. For rare cases of sudden cessation of the heart action, epinephrine should be on hand for intracardiac injection.

DISEASES AFFECTING BOTH HEART AND LUNGS

Only rarely do diseases involve both the heart and lungs simultaneously, and little pause is necessary to consider this association. Miliary tuberculosis may attack the lungs, pleura, pericardium and even the myocardium and endocardium. Rarely may pyemic infections affect the heart and lungs together. Much more frequent associations result from tuberculous or rheumatic infection of the pleura and pericardium, the so-called polyserositis. During the influenza epidemic of 1918, the severest cases showed massive fibrinous involvement of the pleura and pericardium with invasion of the mediastinum. With healing of such processes, there may develop chronic mediastinitis with handicap to the circulation, particularly mediastinopericarditis with resulting chronic obstruction to abdominal circulation causing hepatic cirrhosis and chronic recurrent ascites—so-called Pick's disease.

Inflammation of the superior mediastinum may be tuberculous, syphilitic or pyemic, chiefly as the result of direct extension from inflamed lymphatic glands or abscesses; but inferior mediastinitis appears to be almost always tuberculous or rheumatic, arising from pericarditis or pleuritis. Tuberculosis is in all probability the commonest cause of either type.

THE LUNGS IN DISEASES OF THE HEART

The association of pulmonary symptoms and signs with heart disease is generally widely recognized, but it is profitable to consider the relationship in some detail.

Without doubt the commonest cardiac cause of pulmonary symptoms and signs is congestive failure, no matter what the underlying type of heart disease, whether rheumatic with valve lesions, hypertensive, arteriosclerotic or syphilitic. Dyspnea on ordinary exertion comes first, increasing in degree as the heart fails. This dyspnea, of course, must be due

to the heart disease itself and not to lack of training, effort syndrome, nervous fatigue or pulmonary disease. Certain types of respiratory disturbance may have somewhat different significance than the usual cardiac dyspnea or breathlessness.

Cheyne-Stokes respiration, or marked periodic breathing, is probably the result of faulty cerebral circulation mostly due to weakness of the left ventricle, but favored by cerebral arteriosclerosis. As an event in deep slumber it is not of great significance, but when it occurs during waking hours and particularly if constant, it is a grave sign of circulatory failure.

Orthopnea is generally merely an extreme grade of dyspnea, but when as "cardiac asthma" it occurs paroxysmally at night or day with or without pulmonary edema, it is a serious sign of heart failure, and is even more significant than severe angina pectoris. Its mechanism is not clear, but it is apparently a functional disturbance primarily due to acute failure of the left ventricle with pulmonary stasis and edema resulting from the inability of the left ventricle to take care of the blood sent to it by the relatively normal right ventricle. It is particularly apt to occur with syphilitic aortitis, the failing hypertensive heart and coronary disease (especially thrombosis). It may sometimes be relieved as is angina pectoris by the immediate use of nitrites or alcohol, but it usually requires morphine.

Rapid shallow breathing, tachypnea, is apt to be a nervous symptom or the result of pain on ordinary breathing, while deep breathing, especially with coma, may be a sign of acidosis in uremia or diabetes.

With the development of more advanced congestive heart failure, signs of stasis appear: moist râles at the bases of both lungs and hydrothorax, almost invariably beginning in the right side of the chest and of greater degree there. Some relief of dyspnea may result from tapping such a hydrothorax, but often it will disappear with appropriate therapy, namely, digitalis, rest and diuretics. The hydrothorax of heart failure should not be confused with pleurisy with effusion.

Just as the heart may be displaced by pleural effusions, adhesions, pneumothorax or pulmonary collapse, so too certain conditions affecting the heart, pericardium or great vessels may displace or compress lung tissue, giving rise to unusual symptoms or signs. A heart which is of extreme size may almost fill the left side of the chest and so result in relative dulness over the chest with increase in breath sounds and fremitus, particularly at the angle of the left scapula and in the left axilla. Enormous dilatation of the left auricle has been reported in mitral stenosis, so huge that the signs of involvement of the apex of the left lung or of pleural fluid were found; it is reported that in one such case the right pleural cavity was tapped and the left auricle punctured.

The most common cause for extensive signs of left pulmonary compression, however, is pericardial effusion, not infrequently more extensive posteriorly than anteriorly. It may even be easier to remove pericardial fluid by tapping at the angle of the left scapula than anteriorly or in the axilla; I have seen two such cases. In these patients, x-ray examination is naturally of great value, but the diagnosis should be possible without it, and even in the best of hands the certain diagnosis of pericardial effusion, even though proved extensive by roentgen examination, is not always established. It is not easy at times to distinguish between a very large "mitral heart" and a "pericardial effusion," and I recall one case of effusion that was reported as aneurysm by a roentgenologist.

Aortic aneurysm is another abnormal structure which may displace or compress lung tissue to cause unusual signs. As a rule the diagnosis is easy, the involvement being of the ascending aorta or the beginning of the arch, but if the descending aorta or posterior limb of the arch is involved the signs may be wholly in the left side of the upper part of the chest or the back, and may mislead one to diagnose pulmonary disease. Roentgenograms are, of course, of great value in such cases, which fortunately are rare. One case of pulmonary arteriovenous aneurysm on the right side resulting from a war wound was reported a few years ago by Dr. Marble and myself because of the extraordinary humming top murmur, maximal at the angle of the right scapula.

The only valvular lesion which has particular significance so far as the lungs are concerned is mitral stenosis. There are three complications of importance. The first and commonest is chronic pulmonary engorgement. The veins and arteries are constantly distended and even produce shadows in roentgenograms without there actually being any congestive failure. There usually are no signs of such vascular engorgement on physical examination, but respiratory reserve and vital capacity are both definitely encroached on. Cough with slight blood-stained sputum is not uncommon, especially as the right side of the heart begins to weaken. The second important pulmonary complication of mitral stenosis is the so-called pulmonary apoplexy, often resulting in a brisk and extensive hemorrhage due to the rupture of some pulmonary vessel as a result of the greatly increased intrapulmonary blood pressure. Such hemoptysis has been not rarely confused with the hemoptysis of phthisis, and the patient wrongly treated for tuberculosis. As rest is the essential treatment in either condition, the wrong diagnosis does no harm at first; but to continue to regard the patient with mitral stenosis as tuberculous is, of course, a considerable mistake. The error should not be made, for the differential diagnosis is not difficult. It should be noted that hemoptysis may be the first evidence of mitral stenosis as it sometimes is of phthisis. A third, rather rare, complication of mitral stenosis is

hoarseness due to recurrent involvement of the left laryngeal nerve. The pressure by a large left auricle on the pulmonary artery probably forces the latter against the nerve looped around the aortic arch. Mitral stenosis is, however, much rarer as a factor in the production of recurrent laryngeal paralysis than is aortic aneurysm, nerve disease, tracheobronchial adenopathy and esophageal malignancy.

One further observation about mitral stenosis should be noted, and that is that it is only rarely associated with pulmonary tuberculosis, apparently less often than the law of chance would indicate. It is not known whether or not the explanation lies in the protection of the lungs from tuberculous infection by the chronic hyperemia due to pulmonary vascular engorgement.

There is no particular involvement of the lungs in heart disease due to rheumatism, hypertension, syphilis, hyperthyroidism, angina pectoris or coronary disease unless congestive failure supervenes. With extensive coronary thrombosis, however, blood clot over the infarcted endocardium of the right ventricle or the right auricle may break off to cause pulmonary embolism, just as similar clots may leave the left ventricular endocardium to obstruct some vessel in the brain or elsewhere in the systemic circulation.

COMMENT

I have tried to show that the relationship between the heart and lungs in disease is close both functionally and anatomically, but rather distant etiologically. In order to make correct diagnosis and prognosis and to institute proper treatment, it is necessary to distinguish between cause and effect. This differentiation is usually easy, although occasionally there are obscure cases, and at times both the heart and the lungs are independently diseased in the same patient.

ACQUIRED DEXTROCARDIA *

CARL A. HEDBLOM, M.D.
CHICAGO

In its normal position about one third of the heart lies to the right and two thirds to the left of the midsternal line. In the developmental transposition of the heart and other viscera, or the heart alone, known as congenital dextrocardia, it occupies an isosymmetrical position, two thirds of its bulk being to the right and one third to the left of the midsternal line with the apex in the right nipple line. Acquired displacement of the heart to the right varies from the least discernible amount to the extreme condition in which the heart lies in contact with the lateral or posterolateral thoracic wall, but as a rule its long axis remains unchanged with the apex the farthest to the left. A displacement sufficient to bring about two thirds of the bulk of the heart to the right might by analogy to the congenital type be considered as constituting an acquired dextrocardia. For practical purposes, however, a more definite anatomic delineation is desirable. The left edge of the sternum furnishes a fixed border line for both physical and roentgenologic examination. Acquired dextrocardia, therefore, may be defined as a displacement to such an extent that the whole heart lies to the right of the left sternal border.

Partial displacement to the right is frequently observed, but complete acquired dextrocardia as defined is relatively less common than the congenital type, as judged by the reports of cases in the literature.

Acquired dextrocardia may be due to pressure on the heart and mediastinum exerted from the left, or to a pull from the right. The pull to the right may be exerted directly by contracting bands, attached directly to the pericardium, or by a shrinking lung fixed to the wall of the chest on the one side and to the pericardium on the other, but it also results from the potential vacuum produced by the contracting cicatrizing or atelectatic lung. That the cicatrizing lung may bring about a complete dextrocardia in the absence of pleural adhesions has been frequently observed. In case 4, herewith reported, a dextrocardia exists in association with an extensive pulmonary tuberculosis and a large tuberculous right empyema. Bertier [1] and Bard [2] reported cases in

* From the Surgical Department of the Research and Educational Hospital, University of Illinois Medical School and from the Augustana Hospital.

1. Bertier, J.: Dextrocardie par attraction; pneumothorax thérapeutique. (Contribution à l'étude du mecanisme des dextrocardies acquises), Bull. méd. **27**: 825, 1913.

2. Bard, L.: Du mecanisme de production des désplacements lateraux du coeur, dextrocardies et sinistrocardies acquises, Rev. de méd. **38**:511, 1921.

which an artificial pneumothorax was instituted for the treatment of the dextrocardia. Bard described autopsy observations in cases of dextrocardia without pleural adhesions.

The left lung is typically emphysematous and hypertrophied in these cases of acquired dextrocardia. Instances have been described in which it extended across as far as the right midaxillary line (Everett [3]). Some writers have explained the dextrocardia as due to the crowding exerted by the left lung, but it seems more rational to consider this emphysema and hypertrophy as simply compensatory.

Cicatricial contraction of the lung of sufficient grade to produce an attraction dextrocardia is observed in fibroid phthisis, following pneumonia, particularly of the interstitial type, and bronchiectasis, with associated parenchymal suppuration and sclerosis. Fraenkel [4] mentioned syphilis, malaria, actinomycosis and a "fibroid diathesis" as occasional etiologic factors of a pulmonary fibrosis of this grade. A similar degree of pulmonary collapse is occasionally observed secondary to a massive chronic empyema, but in this condition deformity of the wall of the chest rather than mediastinal displacement is the more marked. I have not found carcinoma of the lung mentioned as a cause of dextrocardia, but I have observed the three cases herewith reported.

A few cases of left pleural effusion with dextrocardia are recorded in which the heart became fixed by adhesions and remained in this position after the effusion cleared up (Chalier and Rabattu,[5] and others).

Pulmonary tuberculosis is the most frequent cause of partial cardiac displacement. Fishberg [6] wrote that in advanced cases it is exceptional to find the heart in its normal place. Of 85 consecutive early cases observed during a period of three months, the heart was displaced to the right in 9 and to the left in 22, and of 29 advanced cases it was displaced to the right in 12 and to the left in 15, but there was a complete dextrocardia in only 5 of 2,344 cases. Oeri [7] reported that in 43 per cent of 413 patients the heart was found displaced to the right and in 82 per cent to the left on admission to the sanitarium.

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3. Everett, W.: Displacement of the Heart: The Apex Beat Below the Angle of the Right Scapula, Brit. M. J. **2**:664, 1900.

4. Fraenkel, Albert: Spezielle Pathologie und Therapie der Lungenkrankheiten; Handbuch für Aerzte und Studierende, Berlin, Urban and Schwarzenberg, 1904, p. 996.

5. Chalier, J., and Rabattu, J. P.: Des dextrocardies acquises, Paris méd. **2**: (Med. Sect.) 365, 1911-1912.

6. Fishberg, Maurice: The Mechanism of Cardiac Displacement in Pulmonary Tuberculosis, Arch. Int. Med. **3**:655 (April) 1914.

7. Oeri, F.: Herzverschiebung bei Lungentuberkulose, Beitr. z. klin. d. Tuberk. **26**:123, 1913.

Seventy per cent of the patients discharged from the sanitarium showed displacement to the right and 97 per cent displacement to the left. Papillon and Flipo [8] recorded two cases of complete dextrocardia in children observed during one year.

Among ten cases of acquired dextrocardia reported herewith, four were due to a right fibroid phthisis, two to a nontuberculous infectious sclerosis, one to a deep roentgen therapy fibrosis and three to carcinoma of the right lung. Four were in males and six in females.

The etiology in sixty cases, including these ten and fifty collected from the literature was as follows: tuberculosis, twenty-eight cases; nontuberculous fibrosis, eleven; trauma with fibrosis, five; bronchiectasis, three; pleuritis, two; carcinoma of the right lung, three, and uncertain, eight, making a total of sixty.

Thirty-six of fifty-six of these cases were in males, and twenty in females. Eighteen of the twenty-eight tuberculous cases were in males and ten in females. Six patients were under 20 years of age —one of these was 7 months—eight were in the twenties and there were three in each of the next three decades. The ages were not given in five cases. The duration of the tuberculosis was from one to five years in five cases and from six to ten years in eight. In three cases, the duration was given as fourteen, fifteen and twenty-three years, respectively. The duration of the dextrocardia was definitely ascertainable in only a few cases; in most of these, it was less than five years.

That a marked attraction displacement of the heart may develop in a short time is indicated by a case reported by Acuna,[9] and others, that of a child, 7 months of age, in which a complete dextrocardia developed in three months.

In addition, the records of ten cases were studied in which disease in the left thorax produced a more or less permanent dextrocardia as follows: tumors of the lung, two cases; diaphragmatic hernia, two cases; eventration of the diaphragm, four cases; deformity of the sternum, two cases.

It is interesting to note that in one case of eventration of the diaphragm that produced dextrocardia, the eventration was presumably due to a neuritis of the phrenic nerve. (There was also evidence of neuritis of the extremities.) The diaphragm eventually resumed its function, as evidenced by the fluoroscope, and the heart then returned to its normal position. It seems possible that similar cases may occur

8. Papillon and Flipo: Dextrocardie acquise par lésion pleuropulmonaire droite, Presse méd. **18**:716, 1920.

9. Acuna, M.; Cibils R. Aguirre, and Galdi, E.: Acquired Dextrocardia in an Infant, Semana méd., Buenos Aires **1**:117, 1927.

following the present day vogue of extraction of the nerve for pulmonary tuberculosis of the left lung.

The heart and mediastinal structures may be displaced to the right en masse without any change in the relative position of the heart, or the heart may be rotated on its axes. The displacement en masse is limited by the points of fixation of the pericardium which are as follows: above, the continuation of the cervical fascia spreading over the great vessels; anteriorly, the superior and inferior sternopericardial ligaments; below, the fixation of the pericardial sac to the central tendon of the diaphragm, and by the vena cava perforating the diaphragm and pericardium. Of these points of attachment, those to the diaphragm are the most fixed. Because of this fixation the heart does not swing over as though the great vessels constituted a hinge as described by Sibson,[10] and others. The direction of the longitudinal axis of the heart typically remains unchanged. In the cadaver the displacement of the pericardium to the right impresses one as being limited, but its relative yielding nature, in some persons at least, is apparent in the sudden dextrocardia from tension pneumothorax and in massive pleural effusions. The extreme displacements observed in chronic cases is no doubt made possible by the prolonged stretching of the points of pericardial attachment.

The massive displacement may be accompanied by a rotation of the heart which is usually on its long axis. Owing to the relationship of the pulmonary artery to the ascending aorta, the rotation clockwise anteriorly from left to right is the least limited. Rotation in the opposite direction is limited by the twist of the right pulmonary artery around the ascending aorta which results. When rotation occurs it is usually clockwise, turning the right ventricle from a right anterolateral to a more anterior position. Rotation due to contracting bands, however, will be in the direction of the attachment and pull of these bands. Babcock [11] reported a case in which necropsy showed the heart twisted from left to right (clockwise) with marked distortion of the great vessels including the inferior vena cava and a second case in which the heart rotated in the opposite direction turning the left ventricle forward and twisting the aorta 90 degrees on its axis.

The most characteristic symptoms of attraction dextrocardia are dyspnea on relative slight exertion, cyanosis and tachycardia. In any given case it is impossible to estimate with any accuracy in what measure

10. Sibson, S.: Malposition of the Heart, in Reynolds' System of Medicine, London, 1877, vol. 4, pp. 125-148; Sibson's Collected Works, London, 1881, vol. 3, p. 204.

11. Babcock, R. H.: A Remarkable Case of Complete Dextrocardia, Med. News **45**:452, 1884.

these symptoms are due to the cardiac displacement, as the same symptoms may be caused by the underlying pulmonary pathologic process. Sauerbruch [12] reported a case of severe dyspnea due to kinking of the trachea. That tachycardia may be definitely due to the circulatory embarrassment from the cardiac displacement is indicated by the fact that it persists in cases in which the pulmonary infection has ceased, the cirrhotic lung representing a healed process as in healed fibroid phthisis. There is also the direct evidence that the tachycardia has been relieved directly following thoracoplastic collapse of the right thorax as in cases 2 and 3 herewith reported. The fact that dyspnea and marked increase in pulse rate is the rule following slight exertion in these cases indicates a small margin of reserve circulatory function.

The outstanding physical signs are cardiac impulse to the right of the sternum, a characteristic marked extensive inflammatory process of the right lung and an emphysematous left lung with absent left cardiac dulness. The cardiac impulse is often strongest in the right axillary line and may be best felt below the lower angle of the right scapula. The right side of the chest lags. Percussion and auscultation yield a variety of signs depending on the pathologic anatomy of the pulmonary process and the amount of associated pleural thickening. The trachea is displaced markedly to the right and may produce signs simulating pulmonary cavitation.

Roentgenographic examination shows the heart shadow to the right with an increased translucency of the emphysematous left lung. The right lung field is decreased, the intercostal spaces narrowed, the ribs slanting more in the direction of the long axis of the body than on the left. If a contrast meal is given, the esophagus is visualized also displaced to the right but the stomach occupies its normal position on the left.

The differential diagnosis between congenital and acquired dextrocardia is based on the position of the abdominal viscera and on the electrocardiogram, the acquired type resulting in normal tracings while the congenital dextrocardia is said always to produce deviations characteristic of that anomaly.

The treatment for acquired dextrocardia on theoretical grounds seems self-evident. If the right lung is so badly scarred that it shrinks to the extreme grade evidenced by the retraction of the mediastinum and the narrowing of the thoracic cavity, the possibility of its ever resuming its respiratory function can be definitely excluded. What more urgent indication can be cited for collapsing the wall of the chest to allow for this inevitable shrinkage and to relieve the tension on the mediastinal structures? If this is accepted in principle, why

12. Sauerbruch: Thorax Chirurgie, vol. 1, p. 286.

should collapsing of the wall of the chest be deferred until a complete dextrocardia has developed? The heart probably as a rule becomes fixed early by adhesions, either to the wall of the chest or to the lung, and therefore cannot be completely restored to its normal position by even the most extensive thoracoplasty. In cases 2 and 3 herewith reported, a complete costatectomy was done, resulting in the greatest possible collapse of the right wall of the chest, but in both cases the heart remains largely to the right of the sternum. A displacement of the heart to the left (sinistra cardia) may produce cardio respiratory symptoms similar to those due to a dextrocardia.

Practically speaking, the various recognized limitations and contraindications to thoracoplasty must be observed. This applied particularly to cases of pulmonary tuberculosis in which the presence of an active lesion in the better lung or elsewhere constitutes possible contraindications. In nontuberculous cases, the general condition of the patient would be the main criterion. In pulmonary carcinoma, thoracoplasty would still serve to meet a temporary mechanical indication and might be combined with exploration with a view to possible radical extirpation.

Pneumothorax has been attempted in a few cases, but such treatment would have to be continued indefinitely without any hope of ultimate restoration of function of the lung.

REPORT OF CASES

CASE 1.—*Unilateral Pulmonary Tuberculosis of Eight Years' Duration with Acquired Dextrocardia.*

C. M., a woman, aged 23, married, seen in consultation with Dr. Stevens of Sioux Falls, S. D., in June, 1925, first developed symptoms of tuberculosis in 1917. In April, 1920, she had a series of pulmonary hemorrhages. Examination at that time was said to have revealed involvement of both apexes. During the next five years which she spent partly at home in South Dakota, partly in Colorado and Arizona, the disease ran an intermittent but progressively downward course, with practically unilateral involvement on the right. There was increasing weakness, dyspnea, tachycardia, cyanosis and weight loss of from 157 pounds (70.8 Kg.) in 1920 to 70 pounds (31.8 Kg.) in 1925. A roentgenogram taken soon after the onset of the illness showed the heart in the normal position. During the latter part of her illness the cardiac impulse was distinctly felt outside the right nipple line. Physical and roentgenologic signs were those of advanced generalized involvement of the right lung with marked retraction of the right thorax and with dextrocardia of a marked degree. The left lung was hyperresonant throughout and did not show evidence of tuberculous involvement.

The patient returned to Arizona in the summer of 1925, where she died in September of that year.

Comment.—This case is a striking example of an extensive persistently unilateral fibrous pulmonary tuberculosis, and the tremendous contractile pull of the scar tissue of the lung. Had the lung been able

to collapse completely there is every likelihood that the disease would have been arrested. Such a case constitutes a striking example of the value of surgical collapse of the wall of the chest in unilateral fibrous phthisis, but such operative collapse must be undertaken before the effect of the disease has become so marked as to preclude surgical relief.

CASE 2.—*Chronic Unilateral Pulmonary Tuberculosis with Acquired Dextrocardia.*

B. G., a woman, aged 31, first developed symptoms of pulmonary tuberculosis in 1919, following an attack of pleurisy. She was admitted to the Municipal Tuberculosis Sanitarium in April, 1924, where the symptoms were those of a moderately advanced right-sided involvement. The sputum was positive for tuberculosis bacilli.

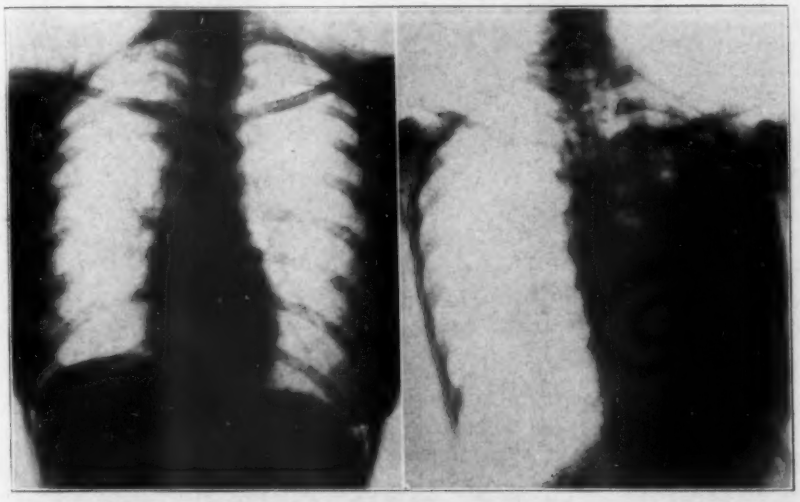

Fig. 1 (case 1).—The figure on the left shows the normal position of the heart at the onset of tuberculosis of the right lung; the figure on the right shows very extensive tuberculosis of the right lung after seven years with complete dextrocardia and deviation of the trachea to the right. There was no roentgenologic or other evidence of tuberculosis in the left lung.

She had lost 15 pounds (6.8 Kg.) in weight during the preceding year. A roentgenogram taken at that time showed the heart partly retracted to the right. The pulse rate was 96; respirations, 28. There was no material improvement during a year's stay at the sanitarium, and an irregular fever persisted with night sweats and the sputum remained positive. In April, 1925, a two-stage thoracoplasty was performed resecting the third to and including the tenth ribs.

The patient was first seen by me in January, 1927. At that time she was dyspneic and there was a marked tachycardia, the pulse rate ranging between 110 and 130. A roentgenogram showed complete dextrocardia. A secondary two-stage thoracoplasty was performed by me at three-week intervals, at which the whole lengths of the previously resected third to eleventh ribs were removed. The ribs

had completely regenerated, and there were massive bridges between each of them. The collapse of the wall of the chest that resulted was much more complete than that which followed the first thoracoplasty. There was marked improvement in the patient's general condition. Two weeks after the completion of the second stage thoracoplasty, the pulse rate had dropped from above 120 to under 100 for the first time in a number of months and the sputum became negative for tuberculosis bacilli for the first time since the patient's admission to the hospital. Her general condition since that time has remained good, but she is weak and has dyspnea on slight exertion.

Comment.—This case illustrates in a definite manner that the tachycardia in cases of chronic tuberculosis with dextrocardia may be due at least in large measure to the malposition of the heart and vessels. In such cases, considerable relief may be expected from

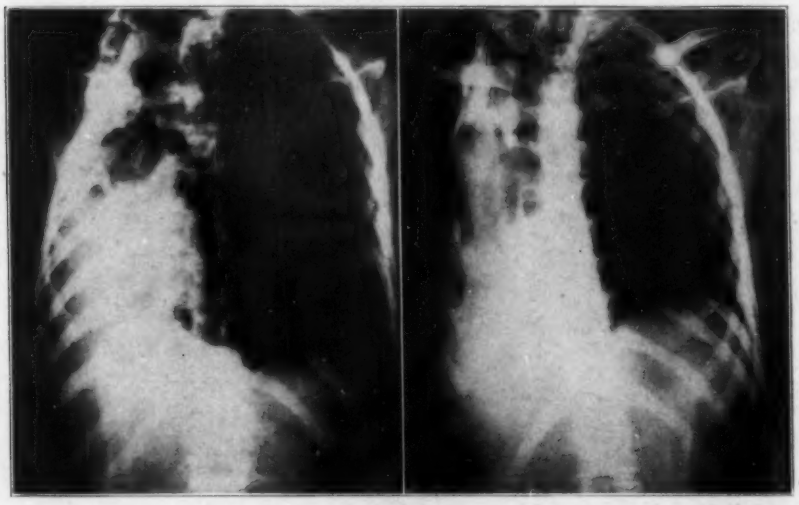

Fig. 2 (case 2).—The figure on the left shows right pulmonary tuberculosis; complete dextrocardia—cardiac impulse in the right posterior axillary line. Partial thoracoplasty was performed elsewhere one year before. The figure on the right shows dextrocardia and deviation of the trachea to the right. The heart persists on the right side after complete costatectomy, but there was marked relief of symtoms particularly of the tachycardia.

thoracoplasty collapse. This case also shows that an incomplete collapse may be insufficient to accomplish this relief. The case further illustrates the fact that long continued progress of the disease results in a certain amount of irreparable damage.

CASE 3.—*Right Unilateral Pulmonary Tuberculosis; Stenosis of the Right Bronchus, Hemoptyses; Thoracoplasty-Pneumolysis.*

B. D., a woman, aged 28, had influenza followed by pneumonia in March, 1919. The pneumonia in her left lung cleared up, but signs persisted in her right lung

and she developed fever, chills and night sweats, with loss of weight and strength. A right pleural effusion developed a few weeks after her illness started. After three months, tubercle bacilli were found in the sputum. She then entered a tuberculosis sanitarium. At this time she was raising about 8 ounces of purulent sputum each twenty-four hours. Artificial pneumothorax was instituted and continued for fifteen months but had to be discontinued on account of advancing adhesions. During this time, she improved considerably and gained 30 pounds (13.6 Kg.) in weight, but she continued to raise sputum and was kept in bed.

After two years of illness, she again began to have fever and developed a complete aphonia. After some months, bronchoscopic examination revealed a stenosed right bronchus. Bronchoscopic dilatation of the bronchus was followed by a greatly increased amount of sputum, decline in fever and marked general improvement. Recurring attacks of chills and fever were relieved by repeated dilatations of the stenosed bronchus. In December, 1923, she had a severe hemoptysis and had many recurring attacks. They seemed to be brought on by lying on the left side and by bending over. The sputum was often blood-stained between attacks. In the autumn, 1924, a bronchoscopic examination was made at the Chevalier Jackson clinic, but she bled so profusely following it that nothing further was done. During the winter of 1924-1925, she had a partial thoracoplasty in stages elsewhere, but the upper three ribs were not resected. During the last month she had spells of fever apparently due to damming up of pus behind the stricture. Profuse hemorrhage recurred at intervals of about a week and she lost 25 pounds (11.3 Kg.) in weight.

She was first seen by me in 1925. She was thin and anemic and slightly dyspneic; the cardiac impulse was palpable over a wide area on the right in the third, fourth and fifth interspaces, but most marked in the midaxillary line. A roentgenogram showed the heart entirely on the right of the sternum. The left lung was hyperresonant throughout. The apex of the right lung was dull to percussion and respiratory fremitus was bronchial. There was an increasing dulness and distant respiratory murmurs toward the base.

A three-stage thoracoplasty resecting the whole length of the upper eight ribs resulted in a satisfactory collapse of the upper part of the lung and was followed by a marked decrease in cough and sputum. The heart also shifted toward the left, but still remained largely on the right. There was, however, a definite decrease in the pulse rate. She had repeated small hemoptyses up to the last thoracoplasty. After discharge from the hospital, she improved gradually. Her temperature fell to normal. Her sputum decreased to 1 or 2 ounces a day and she gained considerable weight. She was troubled less with dyspnea and palpitation. She gradually became ambulatory and was able to go shopping, etc. Because she was still having frequent small hemoptyses although definitely less frequently and less severe than before, she returned to the hospital for another operation.

She entered the Augustana Hospital on July 16, 1926. An x-ray picture taken after the injection of iodized oil 40 per cent showed stenosis of the main bronchus of the right lung. In the hope of accomplishing a better collapse of the lung distal to this stenosis and complete arrest of the hemorrhages, an apical pneumolysis was performed, the lung being freed from the wall of the chest in the region of the stenosed bronchus until there was a cavity between it and ribs about 10 cm. in width and from 6 to 8 cm. in depth. A muscle flap was tucked into the cavity. She recuperated rapidly from the operation. She was discharged home on July 30, fourteen days after the operation.

A later report was to the effect that hemoptyses had been entirely checked although there was occasionally blood-streaked sputum. The patient's general

condition was good. She was free from fever and had gained in weight, but she wrote "I am not very active, but I feel that the great amount of displacement in my chest is what keeps me from being about more on my feet."

Comment.—In this case a persistent hemoptyses constituted an additional indication for thoracoplasty. The first thoracoplasty performed elsewhere was, however, incomplete, nor was the second total costatectomy sufficient to control the hemoptyses. Persistent hemoptyses or an uncollapsed cavity constituted the chief indication for secondary pneumolysis which was effective in this case. This operation incidentally afforded opportunity to explore the pleural cavity which was found adherent. This visceral pleura was studded with healed miliary foci. The case shows further that after the dextrocardia has been present for some

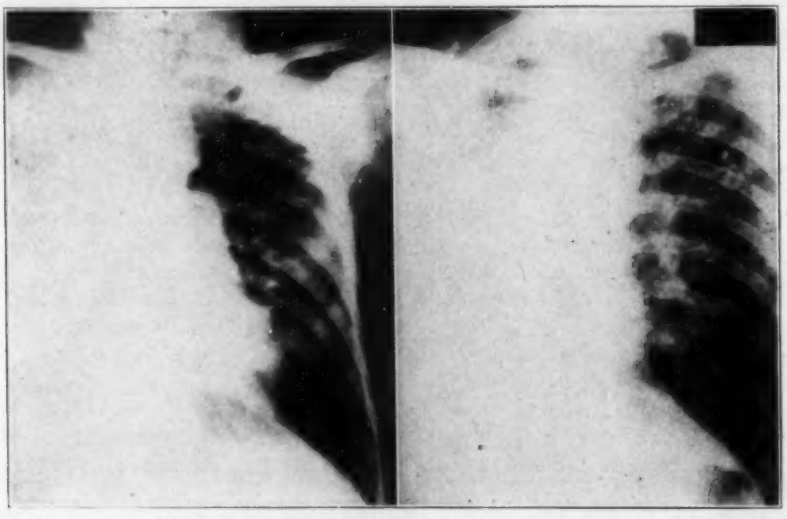

Fig. 3 (case 4).—The figure on the left shows complete dextrocardia in the presence of a large right-sided tuberculous empyema of 2,000 cc. capacity; the figure on the right shows a partial restoration of the heart into the left pleural cavity following complete costatectomy. Marked general improvement was noted.

time the position of the heart may be largely unaffected by even an extensive costatectomy-thoracoplasty. It seems reasonable to believe, however, that the tension on the heart and mediastinum is relieved by such a collapse.

CASE 4.—*Right Unilateral Pulmonary Tuberculosis with Tuberculous Empyema and Acquired Dextrocardia.*

M. B., a physician, aged 48, first developed symptoms of lung infection in 1921. Roentgenograms taken at that time showed tuberculosis. There were no marked symptoms until 1925, when following an attack of influenza he developed fever, cough and sputum, in which tuberculosis bacilli were found. Since that time he has been under treatment, spending most of his time in bed. The sputum varied

in amount up to 4 ounces. The temperature varied from 99 to 100.5 F. Treatment by pneumothorax was begun shortly after the diagnosis of tuberculosis was made and twenty instillations were made, resulting in fairly complete collapse of the lung. Cough and sputum were much decreased, but the pneumothorax was discontinued fourteen months ago on account of pleural effusion. A few weeks before admission, there was an acute exacerbation of fever with a leukocytosis of 17,000.

Examination showed evidence of pleural effusion on the right, the x-ray examination and physical signs being obscured by thickened pleura and fluid. The x-ray picture showed the heart to the right of the left border of the sternum. Exploratory aspiration revealed a purulent effusion which was sterile in culture. The vital capacity was 1,420 cc. About a liter of pus was aspirated, and on March 15 the phrenic nerve was extracted. A three-stage thoracoplasty was begun on March 19, in the course of which the first to the eleventh ribs inclusive were resected. Fluid was drawn from the pleural cavity following each operation until the manometer reading showed a negative pressure. There was no marked change in the position of the heart following the thoracoplasty. The patient was discharged improved, but with a persistent sterile empyema.

Comment.—This case illustrates in a positive way the fact that the shrinkage of the lung is capable of producing a complete dextrocardia even though the lung is not attached to the wall of the chest, as indicated by the fact that an extensive pleural effusion was present in this case. Any material change to the left in the position of the heart is not to be expected as long as there is air in the pleural cavity kept at less than atmospheric tension.

CASE 5.—*Extensive Right Pulmonary Fibrosis with Acquired Dextrocardia Following Extensive X-Ray Treatment After Radical Operation for Carcinoma of the Right Breast.*

Dr. B., a woman, aged 35, entered the Mayo Clinic in March, 1922, because of tumors of the right breast. A diagnosis was made of carcinoma, and a radical amputation was performed on March 28, 1922. The biopsy showed adenocarcinoma without glandular involvement. The patient convalesced satisfactorily from the operation, and on April 10, 11 and 12 was given deep x-ray treatment.

In 1923, she returned complaining of distress throughout the chest, dyspnea, loss of weight and weakness. There was no evidence of recurrence, but examination revealed a pulsation in the right axillary line, suggesting dextrocardia. The roentgenogram showed a thickened pleura on the right and complete dextrocardia. It was the opinion of the consultants who saw her at that time that her symptoms were due to mechanical interference with the heart action. A thoracoplasty was advised, but the patient decided to wait to see if there would be any compensatory relief.

In May, 1924, the patient returned the second time complaining of fever occurring from time to time, usually about every six weeks, and lasting as a rule from eight to ten days. The temperature fluctuated up to 102 and 103 F. She had a chronic cough which was always worse when the fever was present, but which was usually nonproductive. She had pain in the chest and was very weak. She was from 10 to 15 pounds (4.5 to 6.8 Kg.) under weight. The blood pressure was 80 systolic and 60 diastolic as compared with 136 systolic and 80 diastolic in 1922. The vital capacity was 1,390 cc.

Examination at this time revealed a markedly thickened pleura, cavity signs at the right apex, which were thought to be due to a small pneumothorax, and a fibrous lung beneath the trachea pulled into the apical region. The question of tuberculosis was raised, but there was no direct evidence of its presence. A diagnosis of pulmonary fibrosis probably resulting from deep x-ray therapy was made. The patient returned again in 1925, still very weak though she had gained some weight. There was no evidence of recurrence of the malignant disease. She had a complete dextrocardia with the trachea pulled markedly to the right. The left lung was markedly hyperresonant. There was also some edema of the arm, which may have been due to distortion of the vessels.

In September, 1927, the patient reported that she was better, but that her working capacity was reduced on account of dyspnea and lack of endurance.

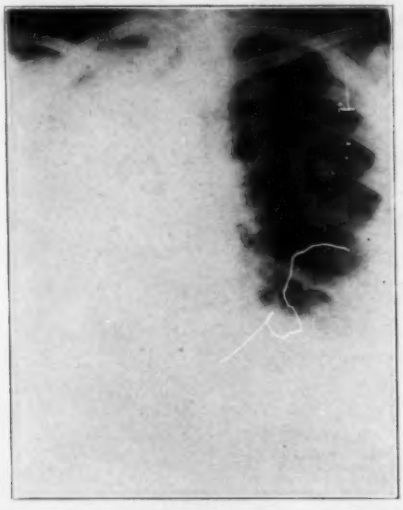

Fig. 4 (case 5).—Chronic pulmonary abscess with a secondary broncheogenic carcinoma; complete secondary dextrocardia.

Comment.—This case represents, as far as I have been able to determine from the available literature, a unique etiologic factor in the development of dextrocardia. The patient's x-ray picture before operation showed the heart in the normal position and there was dextrocardia at the time of her first return in 1923, before she had developed any symptoms suggesting infection. There seems to be no doubt, therefore, that the displacement of the heart is due to a fibrosis of the lung secondary to the x-ray treatment. There is also no other explanation of the dyspnea, tachycardia and distress. What the nature of the process may be which caused the fever later must be left undecided. The rational treatment in a case of this sort would seem to be a thoracoplasty. The relatively low vital capacity would indicate little if any function of the right lung.

CASE 6.—*Postpneumonia Pulmonary Abscess and Fibrosis of Twelve Years' Duration with Acquired Dextrocardia, Acromegalic Changes of the Face and Hands, Without X-Ray or Other Signs of Disease of the Pituitary Gland, Carcinoma of the Bronchus.*

Mrs. F. B., a woman, aged 30, was first seen in May, 1926, because of cough and sputum and pain in the left knee and back. In 1914, she had a severe attack of pneumonia which kept her in the hospital for three months. Exploratory aspiration of the right side of the chest did not reveal any fluid. She developed a cough during this illness which had persisted since that time, accompanied by sputum, varying from 1 to 12 ounces; it was greenish yellow and occasionally blood-streaked, but never foul.

During the last eight months before her admission to the hospital she had six attacks of fever with chills, each lasting ten days. During these attacks she had pain in her hip, left leg and knee. Examination at that time showed signs of consolidation of the upper lobe of the right lung and the heart lying to the right of the sternum. A diagnosis of abscess of the lung was made. Exploratory aspiration in the eighth interspace, posterior axillary line, yielded a large amount of thick gelatinous fluid which was sterile to culture. Open drainage was established, at which time a cavity was opened which was 6 or 8 cc. in diameter and extended very deeply. Her cough and sputum cleared up, and the painful areas in her extremities also disappeared. The sinus continued to discharge a gelatinous mucoid material. After a few weeks, she developed hemoptyses. Injection of iodized oil 40 per cent at this time through the sinus demonstrated that there was a direct communication between the sinus and the bronchus, and also demonstrated an irregular cavity extending upward which showed a connection with the bronchus of the upper lobe. The cough and sputum recurred, and she had several hemoptyses. On this account, it seemed advisable to enlarge the sinus for further drainage. In the course of this exploration, a tumor mass was encountered occupying the lumen of the bronchus of the lower lobe. The bronchus itself was found to be considerably dilated, being perhaps 2 cm. in diameter and completely occluded by the tumor mass. The bronchus was split open with the cautery so that the finger could be inserted beyond the tumor mass which was found to be dumb-bell shaped and about 5 cm. in its longest diameter. Biopsy showed the tumor to be carcinoma. Because of the deep lying position of the tumor and the extensive infiltration at its basal attachment to the bronchus, an attempt to do a lobectomy was abandoned. The patient developed increasing dyspnea and cardiac embarrassment, and died with symptoms of terminal pneumonia. Permission for necropsy could not be obtained.

Comment.—This case represents a pulmonary sclerosis due to a long continued infection of the lung of nontuberculous origin. The dextrocardia possibly may have been due to an atelectatic collapse of the right lung secondary to the development of the bronchogenic carcinoma blocking the bronchus, but this seems unlikely from the fact that the dextrocardia was present when the patient was first observed in May, 1926, while the presence of the tumor was not discovered until about a year and a half later. It would seem most unlikely that a carcinomatous tumor of that size would have been present for such a long period without having shown more extension. In the absence of x-ray and other signs of pituitary diseases, the acromegalic changes were attributed to chronic pulmonary infection.

CASE 7.—*Chronic Postadenoidectomy Pulmonary Suppuration with Acquired Dextrocardia.*

W. B., a boy, aged 6 years, entered the Research and Educational Hospital in March, 1926. An adenoidectomy had been performed, following which the patient developed cough and fever, which was attributed to pneumonia. The cough persisted, but without sputum. He had night sweats and loss of weight. After five months, exploratory aspiration of the chest was done elsewhere and fluid removed. A rib resection was then performed, but pus was not found in the pleural cavity and no further exploration was done. Three weeks later, he began to raise purulent sputum which increased to 8 ounces each twenty-four hours, at times very foul. When first seen by me in March, 1927, he was acutely ill with dyspnea and cyanosis, and there was marked clubbing of the fingers. There were extensive signs of consolidation with cavity formation in the right lung. X-ray examination showed multiple abscesses of the lung with the heart displaced toward the right. A graded drainage operation was performed, first an intercostal incision and later rib resection which was followed by marked improvement. He gained materially in weight and strength, and there was marked decrease in the cough and sputum. He was discharged home in December, 1927, but returned on account of increase in symptoms. In February, he had a severe pulmonary hemorrhage. It was planned to do a graded cautery excision of the diseased portion of the lung. Before this procedure had been started, he developed a severe hemorrhage which resulted in death.

Postmortem examination showed multiple bronchiectatic cavities throughout the right lung with emphysema of the left lung and a dextrocardia.

Comment.—This case is an example of a nontuberculous suppurative process of the lung with marked sclerosis, resulting in dextrocardia.

CASE 8.—*Carcinoma of the Right Lung with Secondary Acquired Dextrocardia and Metastases to the Stomach and Liver.*

Mrs. H. C., aged 37, during the summer of 1924, while residing in South America, developed a slight nonproductive cough which persisted. On her arrival in the United States about three months later, she was repeatedly examined, but evidence of pulmonary infection was not found. At that time an x-ray picture was taken which showed a small circular shadow projecting from above the dome of the right side of the diaphragm, which was suggestive of neoplasm. Cough and sputum continued. She gradually lost weight and strength and began to have almost constant pain in the right side of the chest. The pain was not pleuritic but was localized to the right anterior costal margin. When first seen in May, 1927, she had been having a constant fever up to 100 F. for several months. Examination showed the right side of the chest completely flat to percussion with marked limitation of motion. The heart was drawn to the right beyond the left border of the sternum. There was marked clubbing of the fingers. The sputum had been examined repeatedly by competent observers and no tuberculosis bacilli or other organisms had been found which might account for the pulmonary sclerosis. Resection on the phrenic nerve was performed in May, 1927, which resulted in some relief from the dyspnea and considerably lessening of pain. Frequent examinations were made for glands which might be excised for biopsy, but definitely enlarged glands could not be found. The diagnosis lay between a malignant disease and chronic fibrous tuberculosis. The patient continued to lose in weight and strength, and on August 2, when seen in consultation, she was found to be very much emaciated, dyspneic and cyanotic, with a pulse rate of 130. The right side of the chest was flat to percussion and entirely immobile. There were no palpable

glands. There was a large nodular tumor mass in the left upper quadrant extending to the umbilicus and seemingly adherent to it. The mass was hard with firm edges. The diagnosis was a primary malignant condition of the right lung with abdominal metastases.

Comment.—The diagnosis of the nature of the pulmonary pathologic process causing the dextrocardia in this case must rest on the clinical aspects of the case which, in view of the terminal signs, were as definite as possible.

CASE 9.—*Carcinoma of the Right Lung with Acquired Dextrocardia.*

M. Z., aged 56, entered the Research and Educational Hospital on May 13, 1927. Six months before admission, the patient developed cough with a little blood-tinged sputum. There was gradual and progressive loss of weight and strength, and the sputum became more bloody and very tenacious, and at times

Fig. 5 (case 8).—The figure on the left shows malignant disease of the right lung. The roentgenogram was taken at the onset of symptoms, showing a dense semicircular shadow above the right side of the diaphragm. The figure on the right was taken three years later, showing dextrocardia and deviation of the trachea to the right. Abdominal metastases were first made out a short time before the patient died.

gelatinous. During the last three months, he developed pain in his right side of the chest, and during the last month he lost 12 pounds (5.4 Kg.) in weight.

Examination showed signs of consolidation of the right lung: impaired resonance on the right, especially at the apex and base, with scattered râles and diminished respiratory fremitus. The sputum did not show any tuberculosis bacilli. X-ray examination revealed a marked increase of the hilum shadows, which extended chiefly into the upper lobe, and a heart drawn into the right thorax. The patient died with symptoms suggesting pneumonia.

Comment.—Observations made at necropsy showed atelectatic shrinkage of the lung in addition to fibrous tissue contraction. The atelectasis was presumably due to the blocking of the lumen of the bronchus by the tumor.

CASE 10.—*Right Pulmonary Abscess Secondary to Primary Bronchogenic Carcinoma; Acquired Dextrocardia.*

M. S., a Russian, aged 57, married, a laundryman, entered the Research and Educational Hospital in June, 1927, complaining of cough and purulent expectoration of ten months' duration beginning after a severe cold. The sputum was foul and averaged 10 ounces in twenty-four hours. During the last two months, he had lost 15 pounds in weight. Examination showed marked retraction of the upper right part of the thorax which was markedly dull to percussion. The breath sounds were distant, bronchial and accompanied by crackling râles. Neither tuberculosis bacilli nor shreds of lung tissue could be found in the sputum. A diagnosis of pulmonary abscess, possibly secondary to carcinoma, was made.

Figure 6

Figure 7

Fig. 6 (case 9).—Broncheogenic carcinoma of the right lung with complete dextrocardia. Necropsy showed also an extensive fibrosis and atelectasis of the right lung.

Fig. 7.—Extensive right pulmonary tuberculosis with a complete acquired dextrocardia and marked displacement of the trachea to the right in the presence of a partial right-sided artificial pneumothorax. By courtesy of Dr. Watterson.

An intercostal thoracotomy drainage operation was performed after the pleurae were found adherent and an abscess had been located with the aspirating needle. The patient died the day following operation.

Necropsy revealed an extensive carcinoma of the right lung with secondary abscess formation.

Comment.—The clinical picture presented by this patient was that of a pulmonary abscess, except that the symptoms began without ascertainable cause and his general appearance was suggestive of the cachexia of malignant disease.

EXPERIMENTAL SURGICAL RELIEF OF EXPERIMENTALLY PRODUCED PERICARDIAL ADHESIONS *

ALTON OCHSNER, M.D.
AND
GEORGE R. HERRMANN, M.D.
NEW ORLEANS

The surgical treatment for pericarditis has, in the past, been limited to two stages of the condition: (1) during the acute stage, in the suppurative form, and (2) in the late stage, when an attempt is made to relieve the dense adhesions which impair the cardiac action.

Early surgical intervention is the accepted method of treatment in purulent pericarditis. Surgical treatment is of considerable value in the late stages of the condition, which are characterized by dense adhesions, not only between the two layers of pericardium, but between the pericardium, mediastinal structures and the wall of the chest. The operative procedure of choice in these cases is cardiolysis according to the technic of Brauer.[1] Delorme [2] deserves credit for first having proposed an operative procedure for relief from existing pericardial adhesions. He advocated the removal of the intrapericardial and extrapericardial adhesions in order to relieve the symptoms of cardiac embarrassment. The Delorme procedure was first performed by Rehn,[3] but because of the magnitude of the operation and the almost invariable recurrence of adhesions following their removal, this operation has been more or less abandoned. Parlavecchio [4] first recommended the removal of the pericardium in cases in which there was marked fibrosis of the pericardium, in order to restore the cardiac function. This operative procedure, known as decortication, has recently been revived by Schmieden,[5]

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* From the Departments of Surgery and Medicine, Tulane University Medical School, New Orleans.

1. Brauer, L.: Ueber chronische adhaesive Mediastinoperikarditis und deren Benhandlung, Nat. Hist. Verein. Heidlberg, May 13, 1921; abstr., München. med. Wchnschr., 1922, p. 1072; Verhandl. d. deutsch. Gesellsch. f. Chir. **32**:86, 1903; Arch. f. klin. Chir. **71**:258, 1903-1904; Die Behandlung der Herzbeutelentzuendungen und Verwachsungen, Hamb. med. Ueberseehefte **1**:7, 1914.

2. Delorme: Sur un traitement chirurgical de la symphyse du pericarde, Bull. et mém. de la Soc. de Chir., 1898, p. 827; Gaz. des Hôp., 1913, pp. 86 and 87.

3. Rehn, L.: Zur Chirurgie des Herzens und des Herzbeutels, Verhandl. d. deutsch. Gesellsch. f. Chir. **2**:305, 1907; Zur experimentelle Pathologie des Herzbeutels, Arch. f. klin. Chir. **1**:102, 1914.

4. Parlavecchio: Pericardiectomia sperimentale e sue possibili applicazioni terapeutiche, Policlinico (sez. chir.) **15**:8, 1908.

5. Schmieden, V.: Ueber die Extirpation des Herzbeutels, Zentralbl. f. Chir. **51**:710, 1924; The Technic of Cardiolysis, Surg. Gynec. Obst. **43**:89, 1926.

who, within the past year, has reported eight cases of pericardial "callous" treated by the removal of the markedly thickened and contracted pericardium from the cardiac musculature.

The diagnosis of pericarditis, especially the chronic form, is difficult. Musser and Herrmann,[6] in analyzing the cases of pericarditis in Charity Hospital over a period of five years, found that chronic pericarditis was seldom recognized clinically. In order to determine, if possible, the early signs of chronic pericarditis and also the effect of pericardial adhesions on the heart, in regard to hypertrophy, Herrmann and Musser [7] produced experimental pericarditis in a large number of animals. This experimental study, together with some additional data which we have obtained from observation of our animals, has yielded interesting information.

The classic signs of pericarditis are rarely found in the experimental animal. Retraction of the precordial area was practically never found, except where the pericardium had been sutured to the wall of the chest. None of the neck vein phenomena were observed. On fluoroscopic examination, definite change in the movement of the left border of the heart, especially between the auricle and ventricle, was noted. Electrocardiograms which were taken with the animal in the three positions—on the back, on the left side and on the right side—showed a fixation of the electrical axis, as pointed out by Dieuaide,[8] in some of those cases in which in addition to pericardial adhesions there were dense adhesions in the anterior mediastinum fixing the heart to the anterior wall of the chest. In those cases in which there was no anterior fixation of the heart to the wall of the chest, with a few exceptions, fixation of the electrical axis was not noted. These experimental observations do not, by any means, vitiate the importance of Dieuaide's sign, however, because, as is well known, the dog's mediastinum is not comparable to that of the human being.

Pathologic studies of the material have shown that purulent pericarditis produces little change in the ratio of the weight of the heart to that of the body, as well as the ratio of the left ventricular to the right ventricular weight. The animals survived from three to eight days, which accounts for the small change in the heart. They served as controls for the other animals.

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6. Musser, J. H., and Herrmann, George R.: Chronic Pericarditis: The Clinical Experimental Aspects, J. A. M. A. **87**:459 (Aug. 14) 1926.

7. Herrmann, George R., and Musser, John H.: Experimental Chronic Pericarditis: Further Contribution to the Study of Cardiac Hypertrophy, Proc. Soc. Exper. Biol. & Med. **25**:314, 1928.

8. Dieuaide, F. R.: The Electrocardiogram as an Aid in the Diagnosis of Adhesive Pericardial Mediastonitis, Arch. Int. Med. **35**:362 (March) 1925.

In those animals in which there were external adhesions but no evidence of adhesions between the epicardium and pericardium, the change again was insignificant, aside from a slight tendency toward right ventricular preponderance. This type of chronic lesion of the pericardium of all heart lesions seemed to produce the least amount of cardiac damage.

Animals with a synechia of the pericardium associated with mediastinal adhesions showed a distinct general hypertrophy of the heart. There was a tendency toward right ventricular preponderance in all animals living more than eighty days. In those animals in which the condition had existed the longest period of time, the right ventricle was actually larger than the left.

When there was complete pericardial synechia, with anterior mediastinitis, the greatest degree of general, as well as of right ventricular, hypertrophy was encountered. The pathologic changes were con-

Composite Table: Experimental Pericarditis

	Average	
	L/R	HW/BW
I. Acute purulent pericarditis with mediastinitis	1.279	0.0090
II. Chronic anterior mediastinitis without pericarditis	1.216	0.0092
III. Partial obliterating pericarditis with mediastinitis	1.101	0.0104
IV. Complete obliterating pericarditis with mediastinitis	1.065	0.0116
V. Complete obliterating pericarditis without mediastinitis	1.092	0.0097
V A. Complete obliterating pericarditis without and with slight mediastinitis, second operation	1.130	0.0088

spicuous in all dogs in which the lesion had existed over thirty days and in one dog in which the lesion had been present only seventeen days.

In those animals in which a complete synechia, with no or slight mediastinal adhesion, had existed, and in which the pericardial adhesions had been relieved by pericardotomy, and in a series of animals in which vegetable digestant was added to the pericardium, little change was noticed in the gross hypertrophy.

In a group of eleven dogs with a complete synechia of the pericardium lasting from 14 to 107 days, without any mediastinal adhesions, there was less general hypertrophy than in animals in which there were external adhesions. There was some tendency, however, toward a right ventricular preponderance even in these moderately enlarged hearts.

A summary of the pathologic results obtained in the various series of animals is shown in the table.

OPERATIVE PROCEDURES IN PERICARDITIS

Cardiolysis is of distinct value in those cases of pericarditis in which adhesions between the heart and the wall of the chest exist in addition to the pericardial adhesions. The operation is, however, only palliative

in that it is usually performed in persons presenting marked signs of cardiac embarrassment. Resection of the rigid wall of the chest without any attempt to divide the intrapericardial adhesions, while affording distinct relief to persons with signs of failing compensation, does not cause the cardiac function to return to normal. There is abundant clinical and experimental evidence that when external adhesions are present, internal adhesions are always associated. It also is possible to have complete synechia of the pericardial sac without any evidence of external adhesions. This has been shown repeatedly, both clinically and experimentally. It is logical that an adherent pericardium alone can have a distinct detrimental effect on the function of the cardiac muscle. The adhesions occur most frequently, and are densest, in areas of least movement of the heart. These are the grooves in which the major blood vessels of the heart are located. Owing to the marked fibrosis surrounding the coronary arteries, a decrease in the lumen of the vessel is produced, resulting in an impairment of the nutrition of the myocardium. In cases of synechia of the pericardium, the lubricating effect is also lost.

As we believed that possibly the removal of the pericardial adhesions after the subsidence of the acute symptoms of acute pericarditis might be of value, the following experimental work was undertaken. As has been shown by Rehn, Delorme and others, simple division of the pericardial adhesions is followed almost invariably by the recurrence of adhesions. This property is not only common to the pericardium, but is apparently common to all serous membranes, especially the peritoneum. One of us (A. O.[9]) has been successful in preventing the recurrence of peritoneal adhesions, experimentally, by dividing the adhesions and introducing a digestant into the peritoneal cavity. The fibrin which again forms at the site of the divided adhesions is digested by the digestant before organization takes place; in this way, peritoneal adhesions can be prevented. The results obtained in preventing the recurrence of peritoneal adhesions appeared to justify our employment of digestants in the pericardium. As yet we have not had an opportunity to try the saline solution of the fractional alcoholic precipitate of amniotic fluid as prepared by Shields Warren.

Dogs were used as experimental animals. Before and after each operation and before the animal was killed, an electrocardiographic tracing was made with the animal in three positions—lying on its back, on the left side and on the right side—as well as roentgenograms of the thorax. The latter were taken with the animal on the Stewart[10]

9. Ochsner, Alton, and Mason, Frank: Prevention of Peritoneal Adhesions by Use of Vegetable Ferments, Proc. Soc. Exper. Biol. & Med. **25**:524, 1928.

10. Stewart, Harold J.: A Technique for Measuring X-Ray Photographs of the Cardiac Areas of Dogs, J. Clin. Investigation **3**:475, 1927.

board. Instantaneous exposures were made at the height of expiration. The electrocardiograms were usually taken with the animal under barbital anesthesia, at least for the first two or three times. After the animal became accustomed to the procedure, anesthetization was no longer necessary. No difference was noticed with the animal under barbital anesthesia, except that the sinus arrhythmia disappeared. Fluoroscopic examination was made often for a period of days, especially immediately after the operation. If any unusual symptoms were noted, roentgenograms were taken. The technic of the operation was as follows:

TECHNIC OF OPERATION

Primary Operation with Production of Pericarditis.—With the animal under ether anesthesia, an intratracheal tube was introduced into the trachea, following which ether was administered under positive pressure. Under strict aseptic precautions, an intercostal incision about 5 cm. long was made in the sixth left intercostal space. The skin, superficial fascia and pectoral muscles were divided down to the intercostal muscles. The external intercostal muscle was carefully divided, care being taken not to injure the internal intercostal muscle. Careful hemostasis of the wound was obtained. Because of the thinness of the pleura in the dog, the internal intercostal muscle and pleura were opened together over a distance of about 4 cm. Through the thoractomy wound the pericardium was grasped by means of mosquito forceps, and brought up into the wound. A purse string suture of 00 plain catgut was introduced into the pericardium, following which a small opening was made in the pericardium. Through this opening, irritating inorganic or organic substances were introduced into the pericardial cavity. The pericardium was securely closed by means of the purse string suture, following which the pleura and internal intercostal muscle were carefully closed by means of a 00 plain catgut suture. Accurate apposition of pleura to pleura was obtained in most animals. The wound was closed in layers without drainage. The animal was observed daily until all acute signs had subsided. After varying periods of time, a second pericardotomy was performed, at which time the pericardial adhesions were relieved by digital separation.

Second Pericardotomy.—Again with the animal under positive pressure-ether anesthesia, an intercostal incision was made, usually in the fifth intercostal space, extending from the left lateral border of the sternum to the anterior axillary line. The superficial muscles were divided. The internal intercostal muscle and pleura were divided together. Any evidence of anterior mediastinal adhesions was noted. The pericardium was caught by means of mosquito forceps and delivered into the wound. A transverse incision, about 2 cm. long, was made in the pericardium. In all instances, a complete synechia of the pericardial cavity was encountered. The adhesions were divided largely by means of the finger introduced into the pericardial cavity. At times, however, the adhesions were so dense that sharp dissection became necessary. A marked difference could be noted in the cardiac action before and after dividing the pericardial adhesions. A definite embarassment of the heart's action was demonstrable before division, following which the contraction of the heart muscle became much more forcible. The struggling, tugging, muscular organ, following division of the adhesions, obtained restful release from its fettering bands. In one series of animals, following the digital separation of the adhesions, a vegetable digestant in salt solution was introduced into the peri-

cardial cavity. The pericardium was securely sutured by means of a continuous 00 plain catgut suture. The pleura was carefully closed by means of a continuous plain catgut suture. The adjacent ribs were approximated by means of a chromic catgut suture introduced around the ribs, care being taken not to enter the pleural cavity. The wound was closed in layers without drainage.

Postoperatively, the animals were observed daily until they were killed after varying periods of time. The freeing of the adhesions was borne remarkably well, much better than the production of the pericarditis.

In another series of control animals, physiologic sodium chloride was introduced into the pericardial cavity after division of the adhesions.

In a third series, only digital separation of the adhesions was performed.

At autopsy, evidence of mediastinal adhesions, their character and location, the character of the pericardium and the presence or absence of intrapericardial adhesions were noted. The hearts were removed and fixed; the weight of the body was taken, as well as the weight of the heart. The hearts were divided according to the midseptal [11] and the Lewis technics. The ratio of the weight of the right to the left ventricle was determined. In addition to the heart, the kidneys and spleen were removed. A complete histologic study of the heart and pericardium will be described in a subsequent report.

PROTOCOLS

The following protocols are typical.

Dog 169.—The dog weighed 7.5 Kg.

Dec. 15, 1927: With the dog under barbital anesthesia, after the administration of 1.5 Gm. by stomach tube, electrocardiograms in three positions and a roentgenogram of the heart at a distance of 1.5 meters were taken.

December 16: With the dog under intratracheal ether anesthesia, employing possitive pressure, a midline incision was made over the lower end of the sternum. The pectoral muscles were separated in the midline. Little bleeding was encountered. The sternum was separated in the midline by means of a scalpel and ribshears. After opening the pleural cavity, the pericardium was brought into view by means of forceps. A purse string suture of 00 plain catgut was inserted. A small opening was made in the pericardial sac, and two curetfuls of fused feldspar were introduced into the pericardial cavity. The opening in the pericardial cavity was closed by means of the purse string suture. No attempt was made to suture the pleura. A no. 1 chromic catgut suture was inserted around the sternum, in order to approximate the edges in the midline. On the left side, considerable bleeding was encountered, probably due to injury to the internal mammary artery. The bleeding point was caught and ligated. Before suturing the muscles in the midline, the lungs were expanded by increasing intratracheal pressure. The wound was closed in layers without drainage.

December 17: The animal appeared to be doing well; it lay in the cage, and did not respond readily to external stimuli. Respiration was 44; pulse rate, 120; temperature, 102.7 F. Auscultation of the chest did not show any evidence of friction, either pleural or pericardial. Fluoroscopy showed slight dilatation of the heart. The lung fields were clear.

December 18: The animal appeared weak; it lay quietly in the cage and was unable to stand. The temperature was 102 F.; respiration, 38; pulse rate, 110. The

11. Herrmann, G. R.: Experimental Heart Disease: Methods of Dividing Hearts, with Sectional and Proportional Weights and Ratios for 200 Normal Dogs' Hearts, Am. Heart J. **1**:213, 1925.

breath sounds were somewhat diminished over both sides. Fluoroscopy showed slight enlargement of the cardiac shadow, with a haziness of both lung fields. Attempted aspiration of both pleural cavities yielded nothing. A roentgenogram of the chest is shown in figure 1 *A*.

December 19: The animal appeared livelier and was eating and drinking well. The temperature was 100.5 F.; respiration, 40; pulse rate, 116. The heart sounds were rather distant, but there was no evidence of friction. Fluoroscopy showed the cardiac shadow to be diminished in size.

December 21: The dog apparently was in good condition; it was lively and when allowed out of cage, romped in the room. The temperature was 99 F.; pulse rate, 100; respiration, 30. Fluoroscopy showed only a slight cardiac enlargement with a clear lung field. The wound was in good condition. There was no evidence of suppuration.

December 24: The animal had recovered well from the operative procedure. The wound healed by primary intention. The temperature was 99 F.; pulse rate,

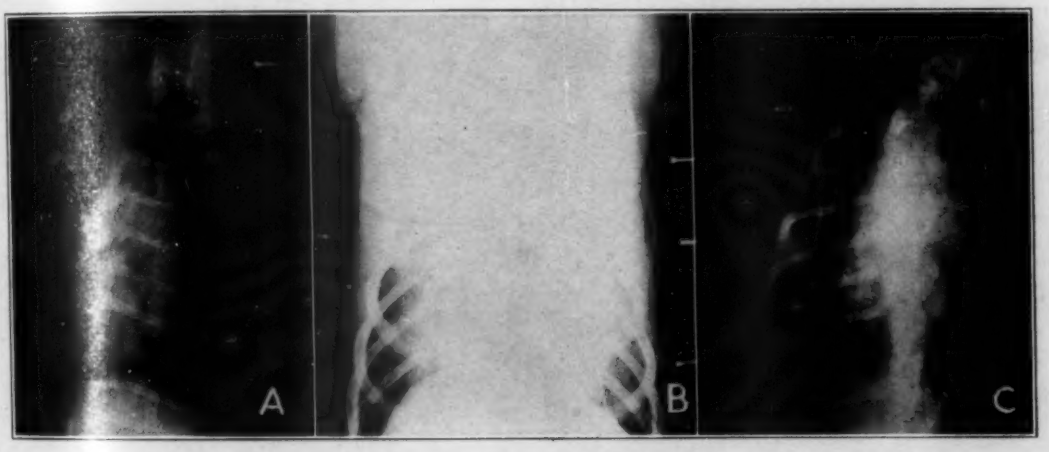

Fig. 1 (dog 169).—*A* shows a roentgenogram of the thorax taken on Dec. 16, 1927, as a preoperative plate; *B*, a roentgenogram of the chest taken on December 18, two days after the production of pericarditis, showing definite enlargement of the cardiac shadow, and *C*, a roentgenogram taken on April 24, 1928, before killing the dog. In *C*, the cardiac shadow has returned to normal.

90; respiration, 24. Fluoroscopy showed a normal cardiac shadow. There was no evidence of a pathologic process in the lungs.

April 1, 1928: Electrocardiograms in three positions and a teleroentgenogram of the heart were taken.

April 3: With the dog under ether anesthesia, intratracheal positive pressure being employed, an intercostal incision was made in the sixth intercostal space, extending from the lateral border of the sternum to the anterior axillary line. The skin, superficial fascia and pectoral muscles were divided. The external intercostal muscle was divided, care being taken not to injure the internal intercostal muscle. The pleura and internal intercostal muscle were opened together for a distance of about 5 cm. After opening the pleura, it was seen that the anterior mediastinum was adherent to the lateral part of the wound, so that one entered the right pleural cavity instead of the left. The pericardium was caught and

brought up into the wound. An opening 2 cm. long was made transversely in the pericardium. There was a complete synechia of the pericardial cavity. The adhesions were densest anteriorly in the region of the original pericardotomy. The cardiac action was visibly embarrassed. A finger was introduced into the pericardial cavity. The adhesions could be broken readily, except anteriorly, where

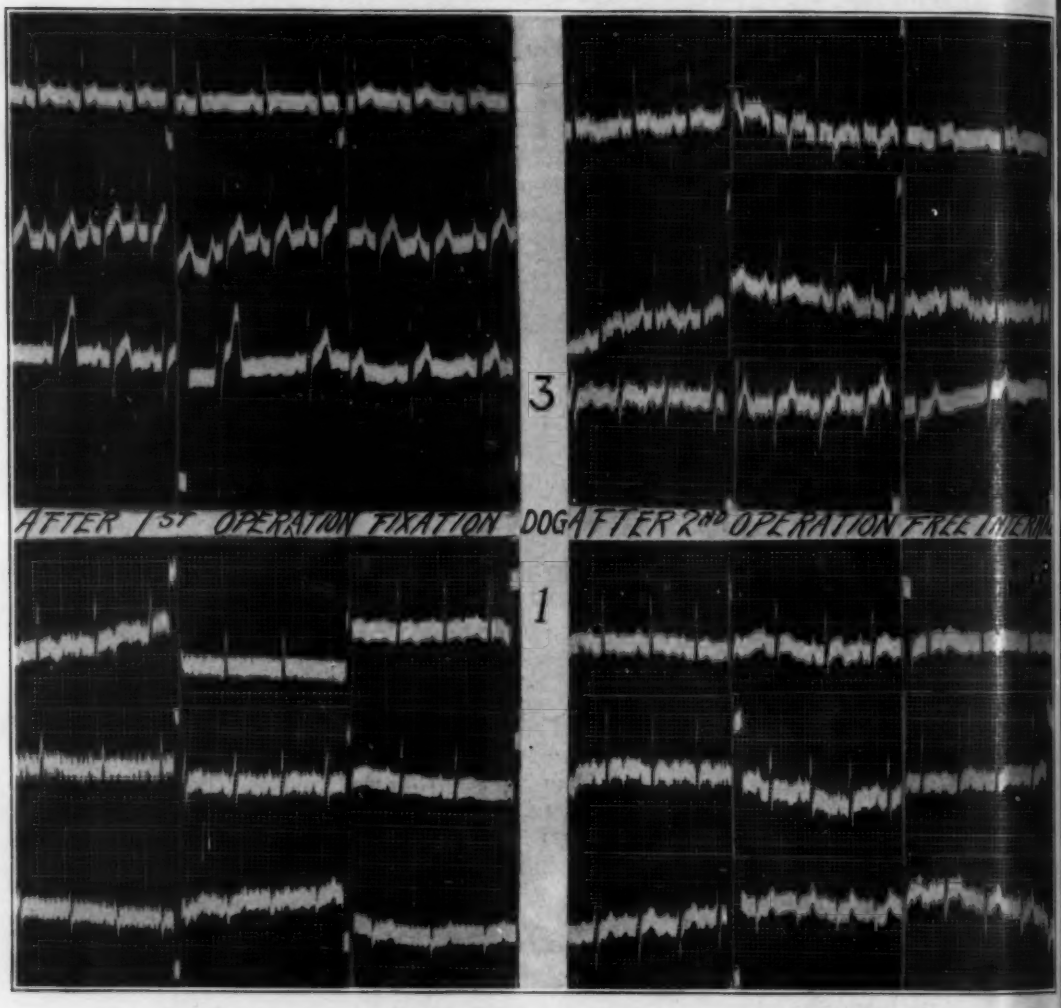

Fig. 2 (dog 169).—Electrocardiogram taken before and after freeing of pericardial adhesions, with the animal on its back, in the left, right and lateral positions. After the first operation, there is fixation of the electrical axis. After the second operation, at which time adhesions were divided, there is no fixation of the electrical axis.

considerable difficulty was encountered. Numerous dense adhesions were found also in the region of the auricles. These were also completely divided. After completely dividing the adhesions, the cardiac action was much less labored. The

pericardial wound was closed by means of a continuous 00 plain catgut suture. Before tying one end of the suture, 50 cc. of a vegetable digestant solution was introduced into the pericardial cavity. The suture was then tied. Two tension sutures of no. 1 chromic catgut were introduced around the ribs above and below the wound, care being taken not to enter the pleural cavity. These were not tied. By means of a continuous 00 plain catgut suture, the pleura and internal intercostal muscles were approximated. At the same time, the tension sutures were tightened, approximating the two ribs. Superficial muscles and fascia were sutured "en masse," by means of a continuous catgut suture. The skin was sutured.

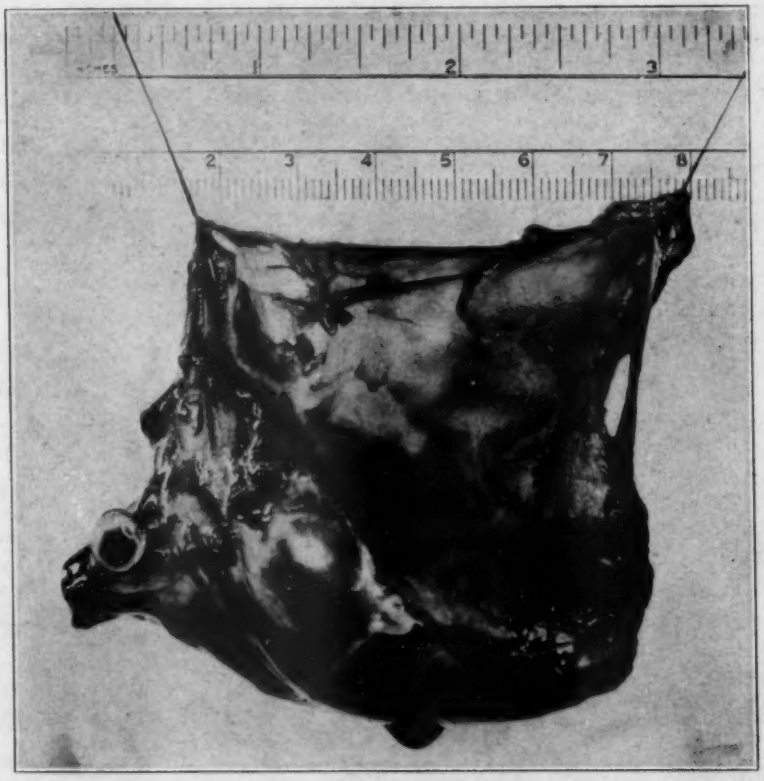

Fig. 3 (dog 169).—The pericardium suspended by sutures. The heart sinks away from the pericardium, showing the nonadherent pericardium suspended above.

April 4: The dog recovered well from the operation with apparently little distress. The temperature was 102 F.; respiration, 38; pulse rate, 132. The results of the examination of the chest were negative. Fluoroscopy showed little change in the cardiac shadow. The lung fields were clear.

April 5: The animal was lively; it ate and drank well. The temperature was 102 F.; respiration, 30; pulse rate, 120. The wound was clean, and there was no evidence of infection. The results of fluoroscopy were negative.

April 6: The condition continued to improve. The temperature was 101 F.; pulse rate, 108; respiration, 24.

April 7: The animal was allowed out of the cage for a few minutes. He was exceptionally lively. The temperature was 99 F.; pulse rate, 98; respiration, 22. The results of fluoroscopy were negative.

April 17: Electrocardiograms in three positions (fig. 2) and teleroentgenograms of the chest were taken. A paradoxic pulse was present. There was systolic retraction of the wall of the chest over the upper precordium. The animal was killed by administering chloroform. Autopsy showed dense adhesions in the right pleural cavity between the pericardium and second thoracic wound. On the left side, the pericardium was adherent to the first thoracic wound through-

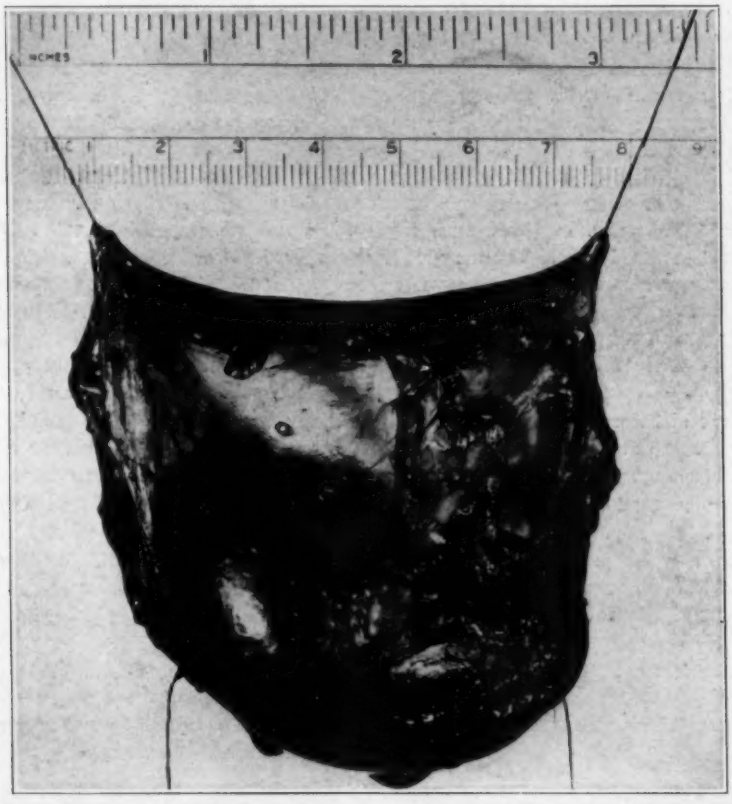

Fig. 4 (dog 169).—Posterior surface of the pericardium suspended, demonstrating the freedom of the pericardium from the adhesions.

out its whole length. The heart was removed without opening the pericardium. The pericardium was apparently free, as it could easily be lifted from the underlying heart. Photographs were taken of the heart with the pericardium suspended (figs. 3 and 4). The pericardium was opened. It was free from any adhesions. Healed scars of the old adhesions were visible, but the pericardial cavity was completely free from bands (fig. 5).

Dog 180.—The dog weighed 11.5 Kg.

Dec. 28, 1927: With the dog under barbital anesthesia, electrocardiograms in three positions and a teleroentgenogram of the chest were taken.

December 30: With the dog under intratracheal, positive pressure, ether anesthesia, an intercostal incision was made in the sixth intercostal space, beginning at the lateral border of the sternum. The pectoral and external intercostal muscles were divided, care being taken not to injure the internal intercostal muscles. An opening was made in the internal intercostal muscles and pleura together. The pericardium was caught by means of mosquito forceps, and brought up into the wound. A purse string suture of 00 plain catgut was introduced into the pericardium. A small opening was made in the pericardium. Four large scoopfuls of fused feldspar were introduced into the pericardial cavity. The opening in the pericardial sac was closed by means of the purse string suture. The pleura and internal intercostal muscles were carefully closed by means of three interrupted plain catgut sutures. This closure was accomplished readily. The superficial

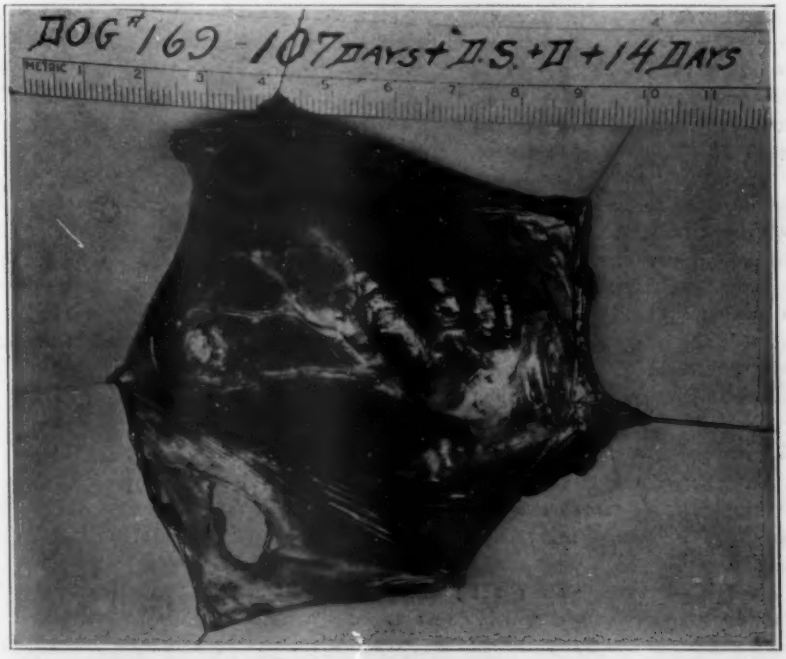

Fig. 5 (dog 169).—Pericardial sac opened; retracted by means of sutures, demonstrating uninvolved pericardial cavity.

wound was closed in layers without drainage. A dressing of compound tincture of benzoin was applied.

December 31: The animal was in good condition. It lay quietly in the cage and ate well. The temperature was 101.2 F.; respiration, 30; pulse rate, 110. The results of the examination of the chest were negative. Fluoroscopic examination showed that the cardiac shadow was slightly enlarged; the lung fields were normal.

Jan. 1, 1928: The animal stood in the cage. It ate and drank well. The temperature was 100.5 F.; respiration, 24; pulse rate, 104. Fluoroscopy of the chest, aside from a slight cardiac enlargement, was negative.

January 2: The temperature was 99.6 F.; pulse rate, 100; respiration, 20. The wound was in good condition. The animal recovered well from the operative procedure.

January 7: Improvement continued. The wound had healed by primary intention. The temperature was 99.5 F.; respiration, 24; pulse rate, 98. Fluoroscopy showed normal observations.

April 4: Electrocardiograms in three positions and a teleroentgenogram were taken.

April 5: With the dog under intratracheal, positive pressure, ether anesthesia, an intercostal incision was made in the left sixth intercostal space just beneath the old scar. The pectoral muscles were divided. The incision extended from the lateral border of the sternum to the anterior axillary line. An opening was made in the pleura and internal intercostal muscles together. There was no evidence of adhesions between the pericardium and the anterior wall of the chest. The pericardium was caught by means of forceps and brought up into the wound. A transverse incision about 3 cm. in length was made in the pericardium. The pericardium was markedly thickened. A complete synechia of the pericardial sac was present. The adhesions were densest anteriorly in the region of the pericardotomy wound. The heart was apparently laboring, due to the adhesions. The adhesions were easily divided bluntly by means of the finger, following which the cardiac action became visibly less hampered. The pericardial wound was closed by means of 00 plain catgut suture. Fifty cubic centimeters of saline solution was introduced into the pericardial cavity. Two stay sutures of no. 1 chromic catgut were inserted above and below the adjacent ribs, care being taken not to enter the pleural cavity. A continuous 00 plain catgut suture, uniting the pleura and internal intercostal muscles, was introduced, following which the stay sutures were tied. A satisfactory closure of the pleura was obtained. The superficial muscles were sutured "en masse." The skin was sutured with continuous catgut.

April 6: The animal recovered well from the operation. It stood in the cage and drank well. The temperature was 102.5 F.; pulse rate, 110; respiration, 32. Fluoroscopy showed some cardiac enlargement, but the lung fields were clear.

April 7: The dog's condition was very satisfactory. The temperature was 101.5 F.; pulse rate, 106; respiration, 34.

April 8: There was a slight fulness of the wound at its anterior extremity, from which some serous exudate was evacuated. The temperature was 101.2 F.; pulse rate, 100; respiration, 36. The results of fluoroscopy of the chest were negative.

April 12: There was a slight purulent discharge at the anterior and posterior extremities of the wound which, however, was apparently very superficial. The temperature was 100 F.; pulse rate, 96; respiration, 30. Fluoroscopy did not show any change in the cardiac or pulmonary field.

April 15: Electrocardiograms in three positions (fig. 6) and a teleroentgenogram were taken. The wound was slightly open at the upper and lower ends. There was a definite paradoxic pulse, but no retraction of the precordium nor any neck vein phenomena. The animal was killed by administering chloroform. Autopsy showed the anterior mediastinum to be free from adhesions (fig. 7). The pericardium was densely adherent to the epicardium, producing a complete synechia of the pericardial cavity (fig. 8). The weight of the animal at death was 11.1 Kg. The kidneys and spleen were removed.

Dog 177.—The dog weighed 8.2 Kg.

Dec. 28, 1927: With the dog under barbital anesthesia, electrocardiograms in three positions and a teleroentgenogram were taken.

December 30: With the dog under intratracheal, positive pressure, ether anesthesia, an intercostal incision was made in the left fifth intercostal space. The

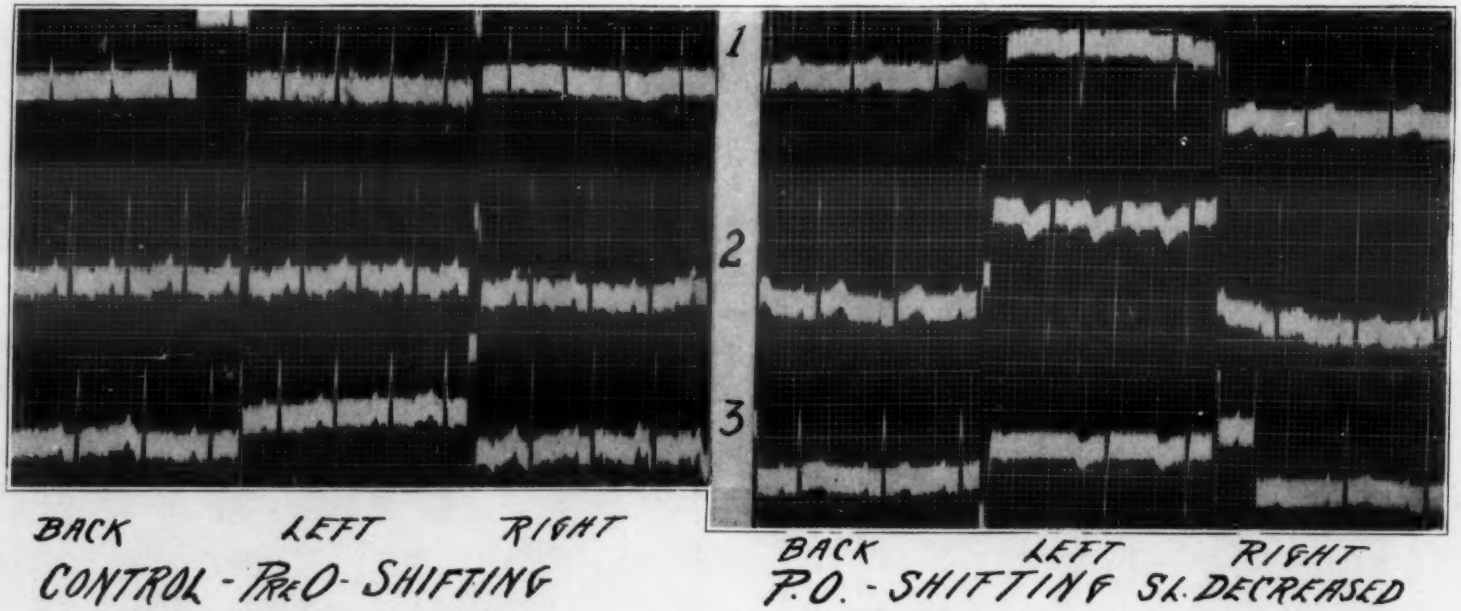

Fig. 6 (dog 180).—Electrocardiogram taken before the second operation in three positions, showing shifting of the electrical axis on the right. Similar tracing taken postoperatively, showing the shifting decreased. The autopsy showed the mediastinum to be free, but the pericardium firmly adherent.

Fig. 7 (dog 180).—Anterior mediastinum with heart in situ, showing freedom of anterior mediastinum from adhesions. The ruler is behind the mediastinum.

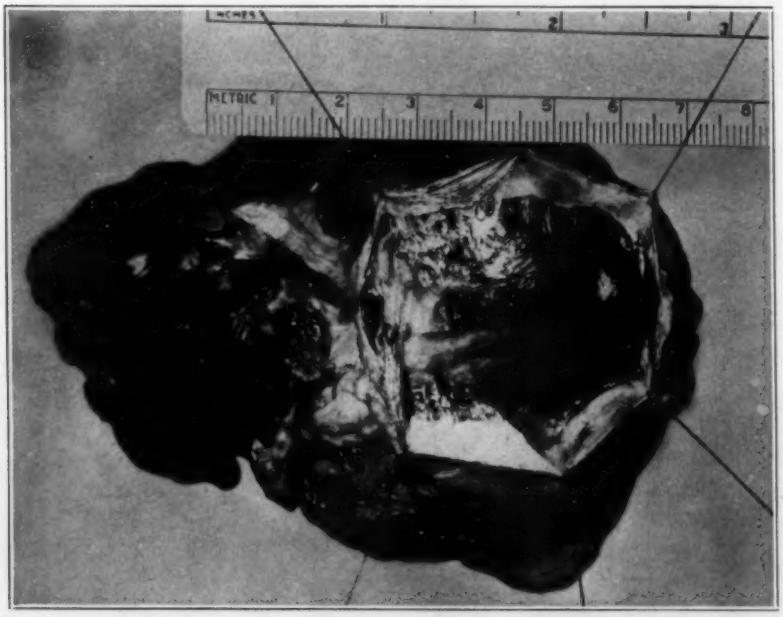

Fig. 8 (dog 180).—Pericardium open, showing dense adhesions between pericardium and epicardium.

pectoral muscles and the external intercostal muscles were divided. The internal intercostal muscles and pleura were opened together. The incision was somewhat too low for a satisfactory approach to the heart, which was located rather high. It was possible, however, to grasp the pericardium by means of forceps, and bring it into the wound. A purse string suture of 00 plain catgut was inserted and the pericardium opened. Three large scoopfuls of fused feldspar were inserted into the pericardium. The pericardial sac was closed by means of the purse string suture. An attempt was made to close the pleural cavity by interrupted plain catgut sutures, which, however, because of the length of the incision, was very difficult. At the upper angle of the incision it was impossible to close the pleura completely. Superficial layers were closed without drainage.

December 31: The dog was up and about and was lively. It apparently was in good condition. The wound was distended. About 1 drachm (3.69 cc.) of sanguineous material was evacuated from the wound. The temperature was 101.8 F.; respiration, 40; pulse rate, 108. Examination of the chest revealed nothing abnormal. Fluoroscopic examination showed the lung fields to be normal and the cardiac shadow only slightly enlarged.

Jan. 1, 1928: There was considerable drainage from the wound, which was becoming slightly purulent. The temperature was 101.6 F.; respiration, 34; pulse rate, 104. The results of fluoroscopy were negative.

January 2: The animal appeared quieter than the day before. The temperature was 102 F.; pulse rate, 110; respiration, 34. The results of fluoroscopy were negative.

January 3: The animal appeared to be sick. It lay quietly in the cage. The wound was discharging some sanguineous, purulent material. The temperature was 102.2 F.; respiration, 30; pulse rate, 104. Fluroscopy showed some cardiac enlargement; the lung fields, however, were normal. The wound was opened and several cubic centimeters of pus evacuated. The wound was filled with a 5 per cent solution of mercurochrome-220. The mercurochrome filling the wounds pulsated with each cardiac beat. No attempt was made to probe the wound to ascertain whether a communication with the free pleural cavity existed or not. Sterile dressing was applied.

January 4: The animal appeared much more lively. The temperature was 101.2 F.; respiration, 24; pulse rate, 96. The wound was draining some at one side; it appeared clean, however. The temperature was 100 F.; respiration, 34; pulse rate, 90.

January 6: The wound was almost completely granulated. The temperature was 99.8; respiration, 30; pulse rate, 90.

March 31: The animal lost considerable weight within the past month. However, it was apparently well otherwise. Under intratracheal, positive pressure, ether anesthesia, incision was made in the fifth intercostal space, extending from the left lateral border of the sternum to the anterior axillary line. The pectoral muscles were divided. The internal intercostal muscles and pleura were opened together. The anterior border of the lower lobe of the left lung was adherent to the old operative scar by three bands of adhesions, which were divided by sharp dissection. Some few adhesions were found between the anterior surface of the pericardium and the old wound. The pericardium was opened by means of a transverse incision about 2 cm. in length. A complete synechia of the pericardial sac was found. Anteriorly, the adhesions were so dense that they cannot be removed bluntly. The cardiac embarrassment was extreme. The finger was introduced into the pericardial sac, and the adhesions were divided bluntly with the exception of those in the region of the old wound, where it was necessary to use sharp dissection.

After freeing the adhesions, the movement of the heart became visibly improved. The pericardial wound was carefully sutured with continuous 00 plain catgut suture. Before tying this suture at its anterior end, 50 cc. of a digestant solution was introduced into the pericardial sac, which filled it completely, but did not produce any apparent cardiac embarrassment. Three stay sutures of no. 1 chromic catgut were introduced above and below the fourth and fifth ribs. The pleura and internal intercostal muscles were sutured by means of a continuous 00 plain catgut suture, following which the stay sutures were tied. The superficial wound was closed in layers without drainage.

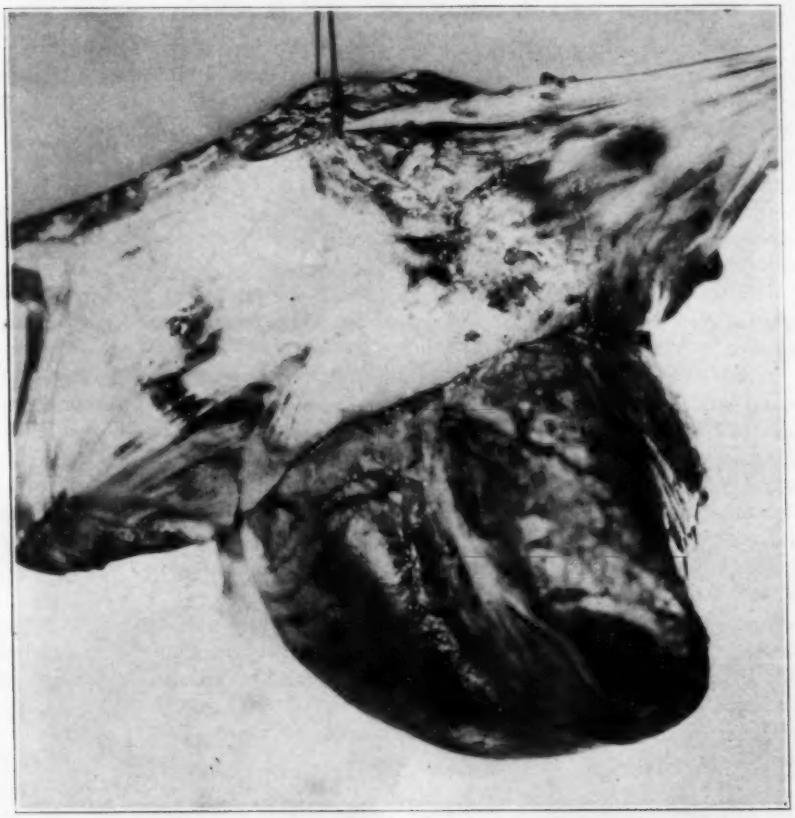

Fig. 9 (dog 177).—Pericardium reflected from heart, showing freedom of pericardium from adhesions.

April 1: The animal apparently recovered from the operative procedure very well. It drank well. The temperature was 101.8 F.; pulse rate, 110; respiration, 30. No friction could be heard. The results of fluoroscopy of the chest were negative.

April 2: The animal's condition apparently had not improved since the day before. There was some swelling in the region of the wound, from which a sanguineous exudate was evacuated. The temperature was 101.8 F.; respiration, 20; pulse rate, 100.

April 3: The animal's condition was not good, but the animal still appeared lively. The wound was opened anteriorly throughout its whole depth. There was an

open pneumothorax, as evidenced by air entering on deep inspiration. Respirations were deep and slow. Respirations were 18; pulse rate, 104; temperature, 102 F. Fluoroscopic examination showed a pneumothorax on the left side.

April 4: The animal's condition was not good. There was still evidence of the pneumothorax. The wound was cleaned and a sterile dressing applied. The respirations were 20; pulse rate, 108; temperature, 101.8 F.

April 5: The animal died sometime during the night. The thoracic wound was wide open. The pleural cavity contained a considerable amount of thick, creamy pus on the left side, extending down to and lying over the pericardial wound. The anterior mediastinum was markedly thickened. The pericardium, however, was free from adhesions, there being no intrapericardial bands (fig. 9).

Dog 202.—The dog weighed 8.4 Kg.

March 13, 1928: With the animal under barbital anesthesia (1.6 Gm.), electrocardiograms in three positions and a teleroentgenogram were taken.

March 15: With the animal under intratracheal, positive pressure, ether anesthesia, an intercostal incision was made in the sixth intercostal space, measuring 8 cm. in length. The pectoral muscles and external intercostal muscles were divided. The internal intercostal muscles and pleura were divided together. Through the pleural wound the pericardium was brought up into the wound by forceps. The pericardium was sutured to the pleural opening by means of interrupted plain 00 catgut sutures. A proteogenous irritant solution was introduced into the pericardial cavity through a small needle, without opening the pericardium. The muscles were closed with continuous plain catgut suture. The skin was closed without drainage.

March 16: The animal appeared very sick. It lay in the cage and would not stand. The respiration was 72; pulse rate, 160; temperature, 102.2 F. Friction rub, more pleural than pericardial, was audible in the region of the heart. The heart sounds were heard well with the animal in a sitting position. With the animal standing, however, the heart sounds were less distinct over the apex. Fluoroscopy showed little change. The cardiac shadow was not enlarged. The heart, however, seemed to be adherent to the anterior wall of the chest on the left side.

March 17: The dog appeared somewhat better than the day before. It drank considerable water, but would not eat. There was marked respiratory embarrassment and spasmodic inspiratory respiration with an expiratory grunt. The pulse rate was 120; respiration, 48; temperature, 104.6 F. The heart sounds could be heard faintly over the wound and distinctly heard around the wound. Water-like râles were heard over the precordium with the animal in a standing position. Friction, mostly respiratory, was heard in the region of the precordium. Fluoroscopic examination did not show any apparent enlargement of the heart. The lung fields were clear. An x-ray plate was taken in expiration.

March 18: The animal seemed to be improving. The respiratory embarrassment was considerably relieved. The pulse rate was 122; temperature, 103 F.; respiration, 44. There was some faintness at the base of the left lung. Fluoroscopy showed slight enlargement of the cardiac shadow.

March 19: There was some purulent exudate in the wound. Respiration was still heavy and labored, with a grunting expiration. The temperature was 101.8 F.; respiration, 26; pulse rate, 110. Fluoroscopic examination showed increased density in the field of the right lung; also an increase in the cardiac shadow. A teleroentgenogram was taken.

March 20: The animal appeared apathetic. It lay quietly in the cage. The wound was distended, and from it a purulent secretion was exuding. The tempera-

ture was 102.2 F.; respiration, 44; pulse rate, 136. One skin suture was removed, and a considerable amount (about 25 cc.) of sanguinopurulent material was evacuated. The fluid seemed to pulsate with each cardiac contraction. It was questionable whether this fluid was coming from the pericardial sac or not.

March 21: There was considerable discharge from the wound. The animal, however, appeared to be better. The temperature was 102 F.; respiration, 40.

March 23: The animal appeared to be sick. It resisted any handling and tried to bite. It did not eat. The temperature was 101.8 F.; pulse rate, 120; respiration, 26. The heart sounds were clearly audible in the standing position, and were heard equally as well with the animal lying on his back. Fluoroscopy of the chest showed a markedly enlarged cardiac shadow. A teleroentgenogram was taken.

March 24: The animal appeared much better. The wound was discharging less and was granulating. The temperature was 100 F.; pulse rate, 110; respiration, 40. It ate and drank well. The heart sounds were clear.

March 25: The animal appeared lively. The temperature was 99.8 F.; pulse rate, 100; respiration, 24.

March 26: Electrocardiograms in three positions and a teleroentgenogram were taken. The animal was in good condition. The wound was completely healed. The results of the fluoroscopic examination of the chest were negative.

March 27: With the animal under intratracheal, positive pressure, ether anesthesia, an intercostal incision was made in the sixth intercostal space, extending from the sternum back to the anterior axillary line. The pectoral muscles and external intercostal muscles were divided carefully down to the internal intercostal muscles. At the anterior extremity of the incision, some dense scar tissue was encountered in the region of the old incision. The pleura and internal intercostal muscles were opened at the posterior end of the incision. The incision was enlarged throughout its whole length. The pleura was firmly adherent to the anterior pericardium, to which it was sutured at the previous operation. The pericardium was caught, just to the left of this attachment, and opened. It was found markedly thickened. On introducing a finger into the pericardial sac, the cavity was found to be completely obliterated by adhesions between the pericardium and epicardium. These could be fairly readily broken by means of the finger, so that all of the adhesions involving the pericardium were freed, with the exception of one very dense strand located anteriorly in the region of the old pericardial wound, where it was necessary to divide the adhesion by sharp dissection. After freeing the adhesions, considerable fluid was found in the pericardial cavity, which was removed by means of an aspirator. There was a great difference noted in the cardiac action after the dividing of the adhesions. The heart's pulsations were much less labored, with a stronger, more forcible contraction. The pericardium was carefully sutured with a continuous plain catgut. Before tying the suture, 50 cc. of a digestant solution was introduced into the pericardial cavity. This solution was sterilized by passing it through a Berkefeld filter. During the sterilization, some water from the water pump sucked back into the solution. It was not known whether a contamination existed or not. It was decided to use the solution, however, and culture it for control. Following the introduction of this material, an attempt was made to suture the pleura, but because of the animal's respiratory movements, it was only partially successful on account of tearing the suture. It was, however, carried out in part. Two interrupted sutures were applied to the periosteum of the ribs, above and below the incision, following which the muscles were sutured, as was also the skin. The length of the operation was forty minutes.

March 28: The animal was in good condition. It was lively and felt playful, and ate and drank well. The temperature was 103.5 F.; pulse rate, 160; respira-

tion, 60. Fluoroscopy showed a normal heart. Culture of the digestant solution injected intrapericardially showed a growth of staphylococci. The heart sounds were clear and regular.

March 29: The animal's condition was improving; it ate well. The temperature was 102.8 F.; pulse rate, 98. A teleroentgenogram showed some cardiac enlargement.

Fig. 10 (dog 202).—The right side of thorax is elevated, in order to show the whole of the pleural wound. Adhesions are seen extending between the pericardium and the original thoroctomy wound, with no adhesions between the pericardium and the second thoroctomy wound.

March 30: The wound was in good condition. The animal's condition was satisfactory. The temperature was 102 F.; pulse rate, 96; respiration, 30. The heart sounds were distinctly audible in a sitting position. There was evidence of a pericardial rub.

April 1: The animal was lively and apparently had recovered well from operation. The temperature was 101.8 F.; pulse rate, 110; respiration, 24.

April 3: The animal's condition was satisfactory. It ate well and was lively. The wound was in good condition. The pulse rate was 120; respiration, 22; temperature, 101.2 F.

April 4: Electrocardiograms in three positions and roentgenograms were taken. The wound was well healed. The pulse was normal, and not paradoxic. There was a slight systolic retraction of the wall of the chest in the region of the first operative scar. The animal was killed by administering chloroform. Autopsy showed several fine adhesions in the left pleural cavity between the anterior border of the lung and the thoracic wound. In the region of the second operative wound, the pericardium was densely adherent over an area 1 cm. in diameter. The pericardium was thickened. The heart was removed. The pericardium was opened. It was adherent to the epicardium posteriorly, but not anteriorly. The spleen and kidneys were removed.

SUMMARY

Sixty-five animals were used in the study. Many were lost as the result of the animals not recovering from the barbital anesthesia after the first operation. In the earlier series, many were lost because of faulty technic. The remainder were again operated on, and the adhesions divided. In one group a digestant was introduced in order to prevent the reformation of adhesions. In another group of control animals, physiologic sodium chloride was introduced into the pericardial cavity. In the third group, merely the division of the adhesions was performed.

Owing to an error in technic in preparing the digestant solutions, which were sterilized by passing them through a Berkefeld filter, on two different occasions the water from the pump was sucked back into the solution, which contaminated the digestant solution. A control culture showed organisms. In the control animals treated by separation of the adhesions, with or without the introduction of saline solution, adhesions invariably reformed. In three of the ten dogs treated by digital separation of the adhesions, with the introduction of a digestant solution into the pericardial cavity, no adhesions reformed. The remaining seven animals showed evidence of extensive pleural infection, with adhesions between the pericardium and the second operative wound. In three, the digestant solution introduced into the pericardial cavity contained organisms. In the three successful cases, extensive adhesions were found and divided at the time of the second operation, and the digestant solution was added. No adhesions were found when the animals were killed two, seven and fourteen days after the second operation.

CONCLUSIONS

Intrapericardial, as well as extrapericardial, adhesions are significant, and produce cardiac embarrassment. Extra pericardial adhesions are usually associated with intrapericardial adhesions. Cardiolysis is only

a palliative procedure to be used in late stages. The ideal time for surgical intervention in pericarditis is during the acute stage of purulent pericarditis, at which time external drainage is indicated. Adhesions, which invariably result, can best be treated after the acute stage has subsided. Evidence is submitted that, experimentally at least, the recurrence of pericardial adhesions following division of such adhesions can be prevented by the introduction of a vegetable digestant into the pericardial cavity. Control animals in which either nothing or saline solution was added to the pericardial cavity following division of the adhesions, these invariably recurred. There is a suggestion that a technic may be worked out which may be of distinct value for treatment in clinical cases of adherent pericarditis. The procedure is of value only after the acute infection has subsided. During the acute stage, adhesions are desirable to limit the infection. The vegetable digestant does not have any effect in the presence of an acute infection.

EXPERIMENTAL PERICARDITIS*

ARTHUR M. SHIPLEY, M.D.
AND
CYRUS F. HORINE, M.D.
BALTIMORE

The pericardium was approached from many different angles. There is no "triangle of safety" in the dog through which the pericardium can be opened without pneumothorax.

EXPERIMENTAL WORK ON ANIMALS

By one method, we resected three or four ribs and pushed the heart over toward the resected area by making pressure on the opposite side. The pericardium was sutured to the wall of the chest in this position with a series of sutures placed in a circular manner. A secondary operation was done several weeks later to open the pericardial sac, which we hoped would be adherent in the area of the previous suturing. This failed in six dogs because the adhesions were pulled out into a thin band, and in each instance the pleura was opened. Other routes of approach were directed through the tendonous portion of the diaphragm and beneath the resected ensiform cartilage. These also were unsatisfactory. We then removed a portion of several ribs, usually the sixth, seventh and eighth on the left side. A purse string suture of heavy linen thread was placed in the intercostal muscle and periosteum. With the purse string suture almost completely tied, a small clamp was introduced within the suture through the wall of the chest, the assistant tying the purse string suture tightly around the clamp. The clamp was passed in an upward direction toward the midline to avoid injury to the lung. By applying pressure on the opposite side of the chest, the heart was forced against the clamp and the pericardium pulled through the opening in the wall of the chest, i. e., the opening surrounded by the purse string suture. In this way we succeeded in opening the pericardium and suturing it to the wall of the chest in twelve dogs. Three of the pericardial fistulas were drained with rubber tubes.

In eighteen dogs, the pericardium was pulled to the outside and infected by puncturing it with a very small hypodermic needle and syringe containing the emulsion of organisms. The pericardium was dropped back into the thoracic cavity. The purse string suture was

* This report is based on observations made on fifty-four dogs. The pathologic changes were almost uniformly the same, and we have, therefore, omitted the details of each experiment to conserve space.

tied, and the wound of the skin closed. The pericardia were all infected with either pneumococci or staphylococci.

In both groups of dogs (the group in which the pericardium was sutured to the skin, including the three cases in which a rubber drainage tube was used, and the group of eighteen in which the pericardia

Fig. 1.—Tube drainage as described in the text.

Fig. 2.—Knife used in the operative procedure. It works in a cylinder. The cylinder contains graphite string packing and is packed around the shaft of the knife by the adjustment screw (*c*). The knife is placed in the incision and the skin clamped between the washers *A* and *A'*. The larger adjustment screw (*B*) is used to tighten the two washers that clamp the skin of the incision.

were infected and dropped back into the thoracic cavity), the post-mortem observations were uniformly the same, i. e., adhesive pericarditis with collections of pus in the auricular areas or lateral recesses, bilateral

empyema and, in many cases, either unilateral or bilateral pneumonia. All of the dogs had adhesive pericarditis involving the apex and a considerable portion of the ventricular area of the heart. In the three

Fig. 3.—Photograph of the window, which is made from the top of a milk bottle. A German glass watch crystal (*A*) is sealed in the mouth of the bottle. The groove (*B*) is made under which the shaft of the knife passes.

Fig. 4.—The window and knife in place. The rays of the arc light pass through the side of the window.

cases in which the tube drainage was used, the parietal pericardium was so firmly attached to the heart that the tube drained only a small localized area of the pericardium rather than the whole pericardial sac (fig. 1).

Beck and Moore,[1] together with other observations, reported the results in a series of dogs in which experimental pericarditis was produced. With the dogs under positive pressure anesthesia, these authors approached the pericardium through the pleura and injected cultures of pneumococcus, nonhemolytic streptococcus and *Staphylococcus aureus*. Some of the dogs recovered with little damage, and a number died within a few days. In our experience, the dogs usually died in from two to three days. Several dogs lived as long as five days after inoculation with the organisms.

Fig. 5.—A turtle's heart before injection of air into the pericardial sac.

As the pathologic changes in the pericardium were so constant, we were interested to know something of the behavior of fluid and air in the normal pericardial sac without an existing artificial pneumothorax. For this experiment two methods were used. The first method was to expose the heart and lungs by removing the sternum and several ribs at the costosternal angle. The triangularis sterni muscles were separated from the pleura. With the use of an arc light, the base of both lungs and the apex of the heart could be seen plainly. The lungs remained

1. Beck, C., and Moore, R.: Significance of Pericardium in Relation to Surgery of the Heart, Arch. Surg. **11**:550 (Oct.) 1925.

in contact with the anterior wall of the chest in expiration as well as in inspiration. The lungs held the heart in close contact with this wall during the respiratory excursions. A needle was passed through the wall of the chest into the pericardium and about 5 cc. of air was injected into it. The base of the heart could not be seen in this experiment. The injected air could not be seen after its introduction into the pericardial sac. The pericardium followed the heart muscle in the complete cardiac cycle as though it were a fixed part of the organ. At

Fig. 6.—The same turtle as in figure 5. The encircled area shows the air in the auricular region.

its base, the heart and pericardium are held more or less fixed by the great vessels, while the pericardium at its lower end is attached to the diaphragm. Six other attempts were made to expose the heart and pericardium by resection of the ensiform cartilage, but we were not able to get another exposure.

Intrapleural and intrapericardial pressures were taken in five dogs. We used a standard large type Harvard manometer. A column of distilled water 30 cm. in height was used. The level of the water was placed on a plane level with the heart. A needle with a slightly curved

point was used in taking the pressures. It was passed through the wall of the chest in the midline near the ensiform cartilage, the point being turned toward the operator when the heart beat was felt against the needle. The needle was held almost at right angles against the heart after the heart beat was felt against it. Slight pressure was exerted on the needle, which was gradually raised and thrust into the pericardium. If the needle passed into the pericardial cavity, the heart could be felt beating against it. If lateral traction were made against the pericardium, the heart could be felt tugging against it. If the needle

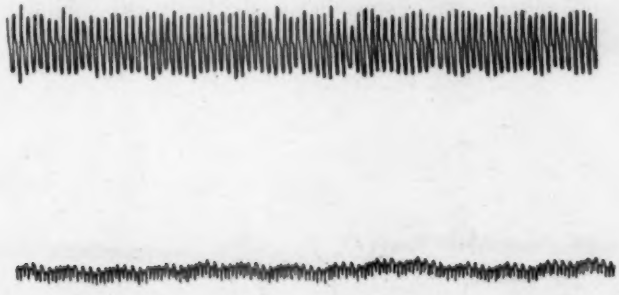

Fig. 7.—Normal tracing before introduction of the needle into the pericardium; systolic pressure, 140 mm.; time in seconds.

became engaged in the heart muscle, tugging against it could be felt immediately. Positive pressure will be transmitted to the manometer if the needle penetrates the heart muscle and enters the ventricular cavities. The pericardial pressures taken in the five dogs showed a pressure of —8, —8, —10, —12 and —14, or an average pressure of approximately —10 mm. of water. Average intrapleural pressures of —140 mm. in inspiration and —50 mm. in expiration were obtained. There was no fluctuation of the column of water with respiration when the pressures were taken within the pericardium. A wide fluctuation of the

column of water will show that the needle is not in the pericardial sac. In each instance in which the intrapericardial pressures were taken the needle was allowed to remain in situ, and section showed the point of the needle within the pericardial sac.

Another method was used to observe the normal heart beat without pneumothorax, by placing a window in the wall of the chest. This was attempted in ten dogs, but we were able to make observations in only two. Extremely exact technic is necessary in order to avoid producing pneumothorax. In the two successful cases, the lung protruded into

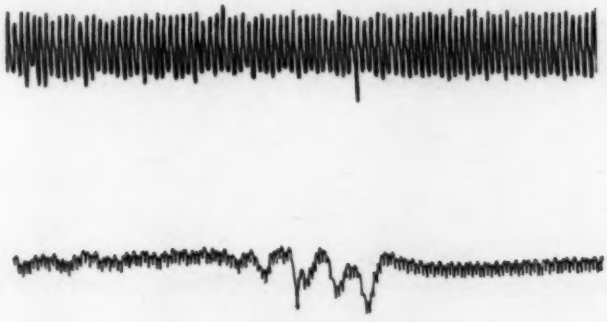

Fig. 8.—Effect of the introduction of the needle into the pericardium. The needle was passed through the wall of the chest in the midline. The needle was passed into the pericardial sac by direct vision of the point of the needle through the window. There was a fall of systolic pressure from 140 to 128 mm. of mercury.

the diaphragm of the window, showing a normal tendency to follow the wall of the chest in the expiratory excursions.

In placing the window, a circular flap of skin is removed on the left side of the chest and a purse string suture is applied in this incision. One or two ribs are resected, and the intercostal vessels ligated. Through another incision several inches away, a special knife is thrust. The knife is shown in figure 2. The window, as shown in figure 3,

is placed in the circular incision and the purse string suture tied. The skin and purse string suture are sealed with flexible collodion. The trapped air between the window and the wall of the chest is aspirated and the puncture wound from the needle is sealed with collodion. An arc light is placed at the side of the window and the heat from the light keeps the lens of the window free from moisture (fig. 4). The point of the knife can then be seen, and the wall of the chest opened. The back of the knife blade is used to retract the lung which comes into the diaphragm of the window and the heart can be observed.

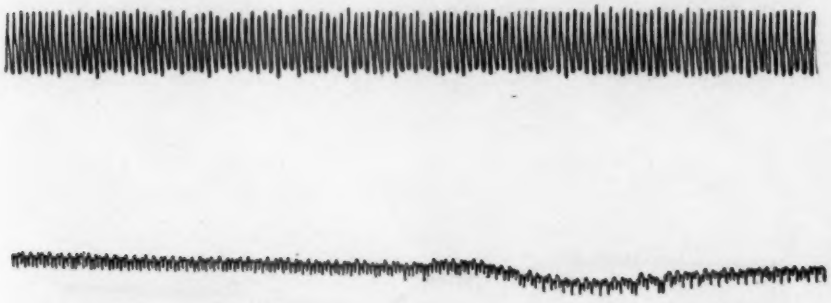

Fig. 9.—Effect of the injection of 10 cc. of liquid petrolatum. The oil was injected at body temperature; time of injection, one-half minute; systolic pressure after injection, 120 mm.

The knife works in a cylinder. The cylinder contains graphite string packing and is packed around the shaft of the knife by the adjustment screw (*c*). The knife is placed in the incision and the skin clamped between the washers *A* and *A'*. The larger adjustment screw, *B*, is used to tighten the two washers that clamp the skin of the incision.

The following observations were made. The pericardium is fixed at the base of the heart by the great vessels and at the apex by a fairly taut tendonous attachment to the diaphragm. The ventricle in systole

moves toward the base line of the heart, as shown by Wiggers.[2] In each cardiac cycle the pericardium follows the movements of the ventricles in systole and diastole as though it were a fixed part of the heart. Small amounts of fluid injected into the pericardium circulated from apex to base and from base to apex. Air injected into the pericardium was forced to the base and remained there, even when the dog was placed in different positions.

We were unable to get photographs of air injected into the pericardium. We therefore tried this experiment in the pericardium of the

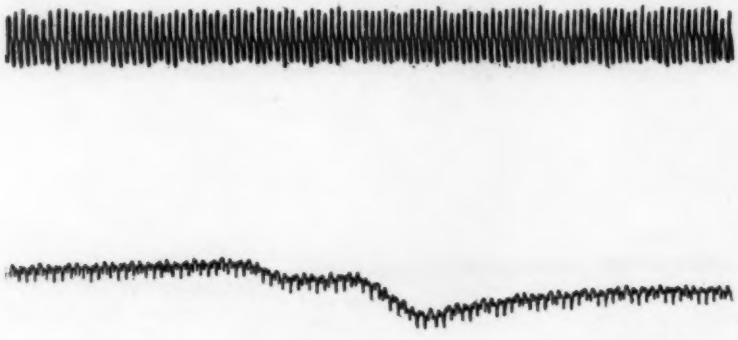

Fig. 10.—Injection of an additional 10 cc. of oil, or a total of 20 cc.; time of injection, one-half minute; systolic pressure before injection, 136 mm.; after injection, 120 mm. of mercury.

turtle. Air injected into the pericardial sac of the turtle's heart gave the same result. When injected into the pericardium, the air is immediately forced to the base of the heart and remains in the auricular area even when the animal is tilted in various positions. The pericardial sac of the turtle in this experiment was exposed to atmospheric pressure.

Tracings were made of the respiratory and pulse curves in the two dogs in which the window was placed, after the injection of liquid

2. Wiggers: Circulation in Health and Disease, Philadelphia, Lea & Febiger, 1923, p. 72.

petrolatum at body temperature into the pericardium. These tracings are shown as observations and are not given as conclusive evidence because of the small series. Katz and Gauchet have clearly demonstrated the effects of the introduction of fluids into the pericardial sac, with special reference to the effects of pressures and pulsus paradosus.

DATA DERIVED FROM CLINICAL OBSERVATIONS

At the meeting of the American Surgical Association in 1927, Dr. Nathan Winslow and one of us (A. M. S.) reported seven cases of

Fig. 11.—After injection of an additional 10 cc. of oil, or a total of 30 cc., into the pericardium; time of injection, one half minute; systolic pressure before injection, 130 mm.; after injection, 114 mm. of mercury.

pyopericardium, and in the ARCHIVES OF SURGERY [3] we reviewed the literature on purulent pericarditis and reported ten new cases, including the seven already mentioned. The experimental data, with our clinical experience, led us to make the following observations.

Operation for pyopericardium is a simple surgical procedure, whether the transsternal or lateral route be used, provided the "triangle

3. Winslow, N., and Shipley, A. M.: Pericardiotomy for Pyopericardium: Review of the Literature to May, 1927, and Report of Ten New Cases, Arch. Surg. **15**:317 (Sept.) 1927.

of safety" be kept in mind. After the pericardium is exposed and incised, however, a number of problems arise in the further treatment that are by no means settled.

There are three widely different factors that bring about the serious consequences in purulent pericarditis—heart tamponage, the infection within the pericardium and the spread of infection to surrounding structure. In a few of the dogs there was a large collection of pus within the pericardium, but most of them showed a relatively small collection. In all of them there was a spreading of infection to surrounding structures.

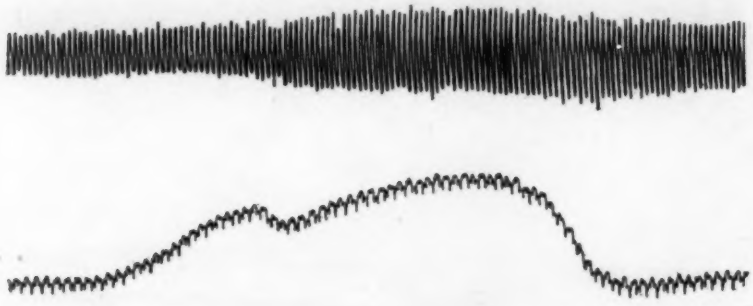

Fig. 12.—Effect of lowering the head of the dog after the injection of 30 cc. of oil. Note the increase in amplitude and rate of respiration. The effect on respiration is the same as will be noted after a total of 60 cc. of oil has been injected as shown in figure 14. Systolic pressure before lowering the head was 136 mm. of mercury. The associated rise in blood pressure will occur in the normal dog. This was done to see what effect the pressure of the fluid might have if all of the fluid be thrown around the auricles. The greatest change has been in the character of the respiration.

The extent of this varied, but this factor was important. In only one of our seven cases of pyopericardium in human beings was the collection of pus very large, and here there was an associated large empyema and the patient died.

There is plenty of clinical evidence that large noninfectious effusions are found within the pericardium provided they accumulate slowly, so it would seem that the infectious process within the pericardium and in the surrounding structures is a more important matter than heart tamponage. Of course, heart tamponage will cause the gravest consequence, if the purulent effusion is large and collects rapidly.

After the pericardium is opened, should drains be introduced? Does their presence increase the number of adhesions? Will the pericardium empty itself if left open, but without the introduction of drains?

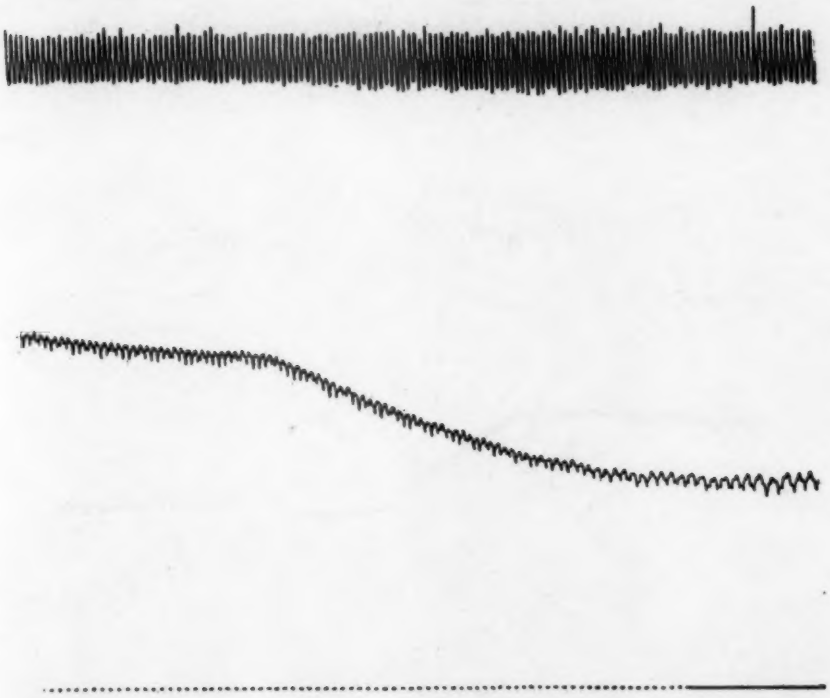

Fig. 13.—Introduction of more oil to a volume of 50 cc. injected; time of injection, one minute; systolic pressure dropped from 117 to 60 mm. of mercury. There was a recovery of the systolic pressure to 90 mm. of mercury. Note the slight increase in amplitude and rate of respiration.

The clinical evidence is confusing, and widely different methods as to drainage have been followed. The postmortem observations in the dogs was disturbing. In every instance there were many adhesions between the two layers of pericardium around the ventricles and pockets of pus found within the pericardium lateral to the auricles at the base of the heart. This condition was about the same whether drains were used or not. When drains were used, they were found entirely sealed off by adhesions and played little part in keeping the pericardium empty.

In human beings, the evidence is more hopeful. In ninety-seven cases in which drainage was used, there were thirty-seven deaths; in thirty-three without drainage, fifteen deaths. Drainage was used in all but one of our seven cases. In this case, the pericardium was stitched to the subcutaneous tissue and left open without the introduction of drainage material. This patient recovered.

In each instance in which the pericardium was opened, there was a gush of fluid, and this phenomenon together with our experience in the last case led us to hope that the action of the heart within the peri-

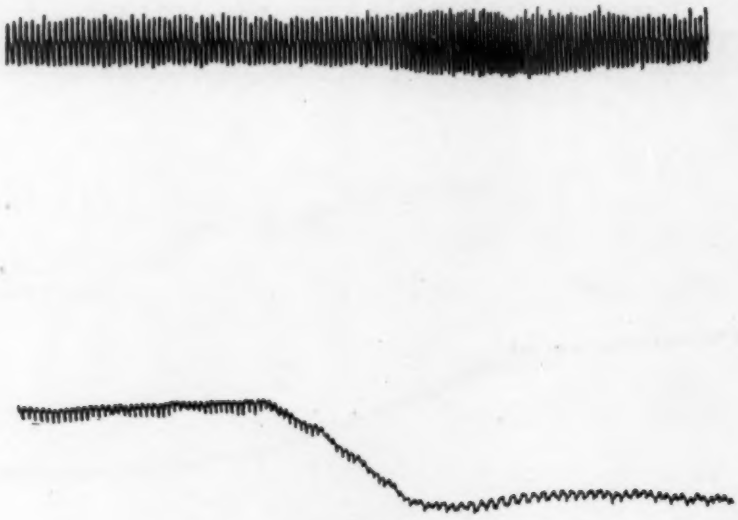

Fig. 14.—An additional 10 cc. of oil was introduced in one-half minute. At this point the oil escaped from the pericardium around the needle. Systolic pressure before injection was 90 mm. of mercury. A total volume of 60 cc. of oil had been injected up to this point. Note the marked rise in amplitude and rate of respiration as was seen after the injection of 30 cc. of oil and lowering the head of the dog as shown in figure 12. Further experiments would be interesting to prove the cause of this respiratory change.

cardium would force out the pus, just as active and passive movement will keep the knee joint drained, but observations in the dogs do not support this belief. With the pericardium freely open and sutured to the pleura, with or without tube drainage, there were adhesions and pus pockets in every instance. In one of our patients there was pocketing following drainage.

After the pericardium is opened, should the finger be carried around the heart in order to break up adhesions and clear up pockets of pus? There are a number of reports of "standstill" of the heart during operation for pyopericardium. In our own cases, the finger was always carried around the heart and fresh adhesions broken up a number of times. In no case did this seriously disturb the heart action. This maneuver was made very gently, however, and care taken not to press or pull on the auricles.

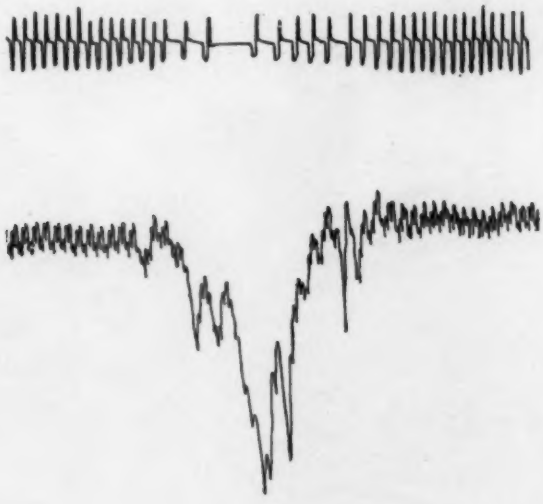

Fig. 15.—Effect of cutting the pericardium. Note the decrease in the respiratory rate. A pneumothorax occurred at this point of the experiment. The needle had been withdrawn from the pericardium. The needle had been in the wall of the chest for several hours. During this time the skin around the needle had been sealed with collodion. Immersion of the dog in water showed that the pneumothorax was produced by air going through the needle puncture wound that remained patent after the removal of the needle. This is noted because of the possibility of formation of pneumothorax in the studies of intrathoracic pressures. The capacity of the pericardium at autopsy was 30 cc.

Should the pericardial sac be irrigated? We did not attempt this in any of the dogs. In two of our patients, irrigation was done with gentian violet solution. Both patients complained bitterly, and in one patient, a youth, aged 19, irrigation was discontinued because of

excruciating pain. However, these two patients recovered. Irrigation, if used, should be safeguarded as to the return flow. Death has been reported during irrigation, and in one instance, death seemed imminent until the removal of the irrigating tube was followed by a gush of fluid with immediate improvement. The temperature of the irrigating fluid should be considered. Physiologists have shown that the temperature of fluids around the heart influences the pulse rate—the warmer the fluid, the higher the rate; the converse also is true. Beck and Moore have shown in one dog that a warm solution, when used as an irrigating fluid, caused adhesions between the heart and pericardium.

Fig. 16.—Heart of experimental dog 12. Note the adhesions of the apex and ventricles.

The postmortem observations on the dogs and the behavior of air in the pericardium revealed a constant tendency to accumulation around the auricles. In one of our patients, there was satisfactory improvement after pericardiotomy for some time when an increasing fever led to investigation. X-ray examination showed an increased shadow to the left of the base of the heart. With the patient under gas anesthesia, a finger was introduced into the pericardium through the drain tract, a number of adhesions were broken up and a pocket of pus was found high up and to the left. The changes in this pericardium closely resembled those found in the dogs.

In the early cases, the fluid was often serosanguineous and the pericardium relatively smooth with little tendency to the formation of adhesions. In the older cases the fluid was thick, the pericardium shaggy and adhesions were the rule.

Our experience with dogs and our observations on patients, together with a survey of the reported cases, cause grave doubt as to the efficiency of drainage as usually practiced for the relief of pyopericardium. There is a tendency for adhesions to form around the ventricles, fluids within the sac are likely to accumulate around the auricles and drains become sealed off. This is especially true in shaggy pericardium with thick pus. If too large an opening is made in the

Figure 17

Figure 18

Fig. 17.—Adhesions in the apical region (*A*) and abscess cavity in the auricular area (*B*).

Fig. 18.—This specimen was taken from a dog that died in twenty-four hours. *A* denotes the apex, *B*, the auricular area. The clouded encircled part of the phtograph shows localized collection of pus, the remaining area is an adhesive pericarditis. This was a constant occurrence.

pericardium, the apex of the heart may be thrust through the opening and compressed. One of us (C. F. H.) suggests the possibility of trephining the sternum, over both the base and the apex and making two small openings in the pericardium, one at the top and one at the bottom. We were unable to devise any method of doing this experimentally in dogs.

Both the clinical and the experimental evidence show that there is a tendency for pockets of pus to collect lateral to the auricles. The usual

opening in the pericardium over the apex of the heart does not always bring about adequate drainage. Just what might be done to improve drainage around the auricles is a problem of importance.

In purulent pericarditis with a moderate amount of pus present, it is likely that pressure on the ventricles is not very disturbing to the circulation, but it is known that pressure around the base of the heart interferes with the filling of the auricles, thereby raising the pressure in the veins and lowering it in the arteries. It would be interesting to know what effect the bathing of the heart in pus has on the sino-auricular node and the auriculoventricular bundle.

There has been considerable controversy as to the position of the heart within the distended pericardium in purulent pericarditis. Three different opinions have been held. One is that the heart is pushed back away from the sternum; another that the heart is surrounded on all sides by a wall of fluid, and the third belief is that the heart lies as it does normally with the base pointing obliquely backward and the apex obliquely forward. Our observations coincide with the latter view. In the patients operated on, the apex was always found against the anterior pericardium, and at first this was a disturbing observation as it caused doubt as to the presence of fluid within the sac.

So many cases of pyopericardium are overlooked that it raises the question whether it is not possible to have a serious disturbance of the circulation by an amount of pus within the pericardium too small to show any marked increase in the pericardial shadow or to give any clear physical signs of its presence. Holmes [4] emphasized the use of the fluoroscope in pericarditis and expressed the belief that a pathognomonic sign is inability to distinguish the auricular from the ventricular beat.

Because the pressure everywhere within the thorax, except in the lung itself, is lower than atmosphere, one's curiosity is raised as to the effect of atmospheric pressure on the heart after pericardiotomy. Certainly the function of the wall of the chest is to maintain a negative pressure within the thorax, and it is not unreasonable to suspect that the heart may be disturbed by exposure to atmospheric pressure. We believe that closed drainage of the pericardium in purulent pericarditis might lessen some of the imperfect results following on drainage.

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4. Holmes, G. W.: Use of Roentgen Ray in Diagnosis of Pericarditis, J. A. M. A. **83**:1745 (Nov. 29) 1924.

THE PRESENT STATUS OF THE SURGICAL PROCEDURES IN CHRONIC VALVULAR DISEASE OF THE HEART

FINAL REPORT OF ALL SURGICAL CASES *

ELLIOTT C. CUTLER, M.D.
AND
CLAUDE S. BECK, M.D.
CLEVELAND

Because the major topic for discussion at the meeting this year is the heart it seems opportune to review the cases of valvular disease in which surgical treatment has been used. In summarizing these cases, we shall attempt to evaluate the general idea of subjecting such disorders to surgical therapy and we shall also attempt to emphasize the problems that must in the future be overcome to make surgical procedures on the cardiac valves useful and beneficial.

Operation has been performed in twelve cases of chronic valvular disease of the heart. These cases have been reported in detail except the last two in which we performed the operation. For the details of these procedures, the reader is referred to the original reports.

SUMMARY OF CASE REPORTS

The table shows, in chronological order, the cases in which operation has been performed.

CASE 1 (Doyen [1]).—A woman, aged 20, had a condition which was diagnosed congenital pulmonary stenosis. The chest was opened and the heart exposed. A small tenotome knife was inserted into the right ventricle and an attempt was made to divide the stenotic valve. Death occurred several hours after operation. At autopsy, there was found a narrowing of the conus arteriosus rather than a localized stenosis of the valve. In addition, and as is usual in cases of congenital disease of the heart, other defects were present, viz., perforated interventricular septum (Roger's disease) and incomplete development of the lungs.

CASE 2 (Tuffier [2]).—A young man showed signs of a marked and progressive aortic stenosis. The thorax was opened, and the root of the aorta was exposed. It

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* From the Department of Surgery of the Western Reserve University School of Medicine and the Lakeside Hospital.

1. Doyen, E.: Chirurgie des malformations congenitales ou acquises du coeur, 26th Cong. de l'assoc. franç. de chir., Presse méd. **21**:860, 1913; La chirurgie du coeur et des vaisseaux, Soc. d. l'inter. d. hôp de Paris, ibid. **21**:987, 1913; La chirurgie du coeur et des gros vaisseaux, Soc. de l'inter. d. hôp. de Paris, ibid. **22**: 282, 1914.

2. Tuffier, T.: État actuel de la chirurgie intrathoracique, Tr. Internat. Cong. Med., 1913, London, 1914, Sect. 7, Surgery, pt. 2, p. 249; discussion, p. 326, 1914; La Chirurgie du coeur, Cinquiéme congrés de la Société internationale de chirurgie, Paris, July 19-23, 1920, Rapports Procés-Verbaux et Discussions, publiés par le Docteur L. Mayer, Brussels, Hayez, 1921, pp. 5-75.

was the intention of the operator to insert a knife above the aortic ring and to incise the stenosed valve, but the procedure was changed to a dilatation of the aortic ring by invaginating the wall of the aorta just above the valve and pushing the wall into the stenosis on the fore-finger. As late as 1924, this patient was reported living and improved.

CASE 3 (Cutler and Levine[3]).—A girl, aged 11, did not give any history of acute rheumatic fever. There was dyspnea on exertion for three years. For eight months previous to operation, she was confined to bed, and during this period had alarming attacks of hemoptysis. A diagnosis of mitral stenosis was made. A

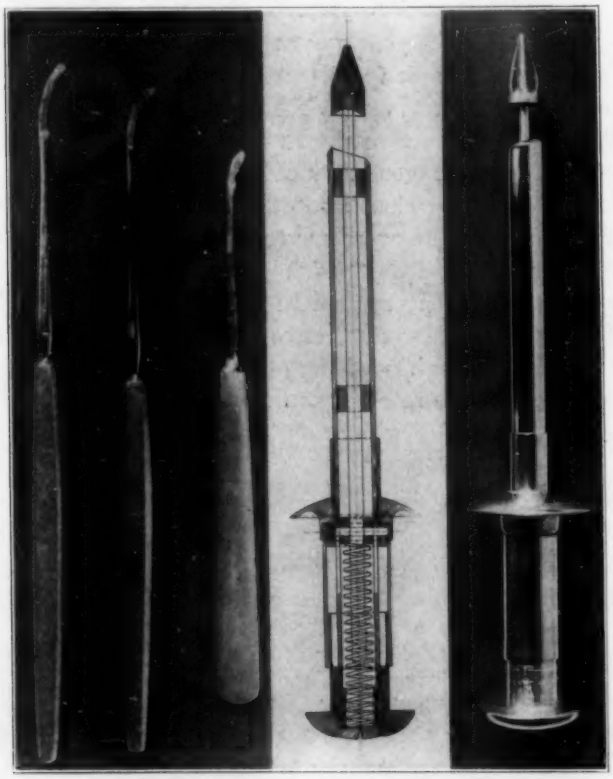

Fig. 1.—Tenotome knives and cardiovalvulotome. The former were used in cases 3, 5 and 6; the later was used in cases 7, 8, 10, 11 and 12.

roentgenogram showed that the left auricle was prominent. The patient was anesthetized with ether, and the chest was opened by a median thoraco-abdominal incision. A tenotome knife (fig. 1) was inserted into the left ventricle, and an attempt was made to incise each cusp of the obstructing ring. The wound in the heart was sutured with silk. The pericardium was closed tightly without drainage.

3. Cutler, E. C., and Levine, S. A.: Cardiotomy and Valvulotomy for Mitral Stenosis, Boston M. & S. J. **188**:1023, 1923. Cutler, E. C.; Levine, S. A., and Beck, C. S.: The Surgical Treatment of Mitral Stenosis, Experimental and Clinical Studies, Arch. Surg. **9**:689 (Nov.) 1924.

Fig. 2 (case 3).—The mitral valve as seen from the auricle.

Fig. 3 (case 3).—The mitral valve as seen from the ventricle. The site of the incision is indicated.

An effusion developed in the pericardial cavity during the first three days after operation producing heart-tamponade, and the condition of the patient was critical. With absorption of the fluid, however, the condition improved. Signs of mitral stenosis persisted; the diastolic murmur was somewhat changed and there was some prolongation of the systolic murmur. The patient lived four and a half years following the operation, and during this period her activities were restricted; on several occasions, she had to be admitted to the hospital for rest in bed. The terminal illness was abrupt. Dyspnea increased; a hacking cough developed; the temperature rose to 103 F.; there were râles at each base; there was no peripheral edema. The dyspnea became severe, and the patient died on Nov. 17, 1927.

Necropsy showed a marked anterior bulge of the sternum and thoracic wall. The longitudinal incision in the sternum was well healed. The pericardium was adherent to the sternum. The pericardial cavity was obliterated by generalized adhesions between the heart and pericardium. The heart was markedly enlarged. Both auricles were capacious, but the left was markedly enlarged and could contain easily a good sized fist. The mitral valve was moderately stenosed and thickened. The other valves were normal. A bougie, 40 mm. in circumference, readily passed through the mitral orifice (figs. 2 and 3). The site of the incision made in the mitral valve at operation was easily determined. It was at the anterior junction of the aortic and posterolateral cusps. There is no doubt that the orifice was enlarged by this incision. The scar in the ventricle was well healed. The lungs were markedly congested and showed some bronchopneumonia.

Case 4 (Allen and Graham [4]).—A woman, aged 32, did not give any history of rheumatic fever. Dyspnea had been present for many years and hemoptysis for one year. There were orthopnea, distressing cough, edema of the ankles and cyanosis. A presystolic murmur with a thrill was present, also a systolic murmur. The heart was enlarged. The systolic blood pressure was 95 mm. of mercury, and the vital capacity was reduced. A diagnosis of mitral stenosis was made. The operation was carried out in three stages. At the first stage the costal cartilages of the first, second and third ribs were removed under procaine hydrochloride anesthesia. At the second stage, the incision was opened under nitrous oxide-oxygen anesthesia and the pericardium was exposed. The pleura was opened, but because the patient's respirations became embarrassed the operation was discontinued. At the third operation, the incision was reopened under gas-oxygen anesthesia. The pericardium contained 500 cc. of clear fluid. The left auricular appendix was distended. There was no thrombus in it. The cardioscope was inserted into the left auricle. At this stage in the operation respirations, which were greatly embarrassed, ceased and the heart stopped beating. Epinephrine was injected into the heart, but the patient died.

Case 5 (Cutler, Levine and Beck [5]).—A woman, aged 35, did not give any history of rheumatic fever. Dyspnea on exertion and precordial pain were present for four years, and these symptoms required frequent hospital care. Edema of the ankles, enlargement of the liver and auricular fibrillations developed. The patient was bedridden. The systolic blood pressure was 85 mm. of mercury preceding operation. A diagnosis of mitral stenosis was made. A median thoraco-abdominal incision was carried out under ether anesthesia. The auricle was a huge, pulseless sac of blood. Three attempts were made to incise the stenosed mitral valve with the tenotome knife inserted through the left ventricle. After the third attempt the

4. Allen, D. S., and Graham, E. A.: Intracardiac Surgery, a New Method, J. A. M. A. **79**:1028 (Sept. 23) 1922.

5. Cutler: Levine and Beck (footnote 3, second reference).

heart stopped momentarily, but almost immediately began at the rate of 50. The immediate postoperative recovery seemed good, but the circulation gradually failed and death occurred ten hours later from myocardial failure. Necropsy examination showed the mitral valve to have been only slightly enlarged (fig. 4).

CASE 6 (Cutler, Levine and Beck[8]).—A man, aged 26, presented a history of rheumatic fever in childhood. Dyspnea and substernal pain were the first symptoms. There was a diastolic and presystolic murmur with a thrill. The second sound was accentuated. The systolic blood pressure was 105. The vital capacity

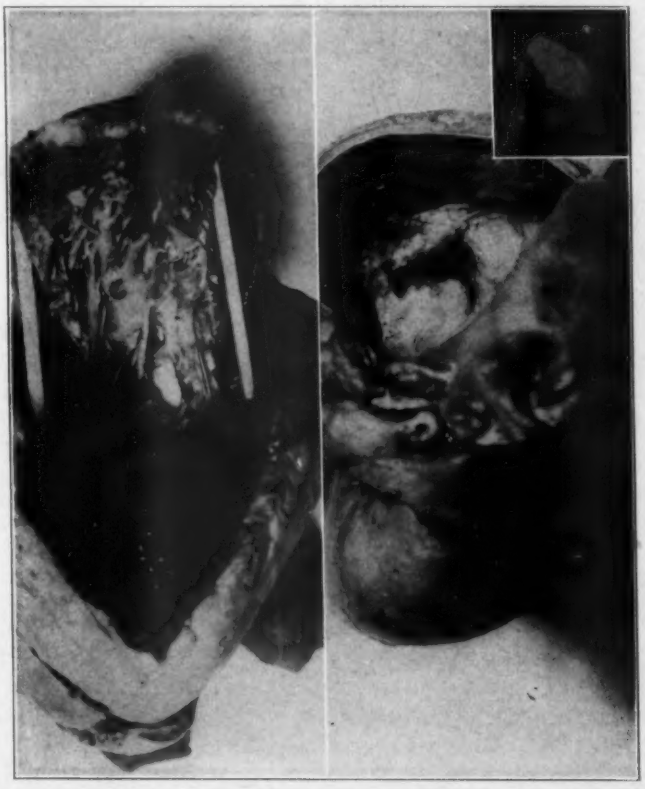

Fig. 4 (case 5).—Auricular and ventricular views of the mitral orifice; the roentgen-ray film (inset) shows some calcium deposition. The sharp border of the valve edge in the ventricular view where the incision was made at operation may be noted.

was 72 per cent of normal. A diagnosis of mitral stenosis was made. Under ether anesthesia, the operation was carried out. The chest was opened by a median thoraco-abdominal exposure. Extensive fibrous adhesions between the heart and the pericardium were cut. Two attempts were made with the tenotome knife through the left ventricle to incise the calcareous mitral ring. The pericardium was closed tightly without drainage. The procedure was well tolerated. During the postoperative period which lasted twenty hours, the patient presented the picture of a failing circulation. At necropsy, 350 cc. of fluid were found in the pericardial

Fig. 5 (case 6).—*A* shows the ventricular aspect of the heart; pericardial tags and roughness may be noted. The mitral orifice shows divided chordae tendineae and slight notching at either end of the long diameter made by the knife at operation. *B* shows the auricular aspect.

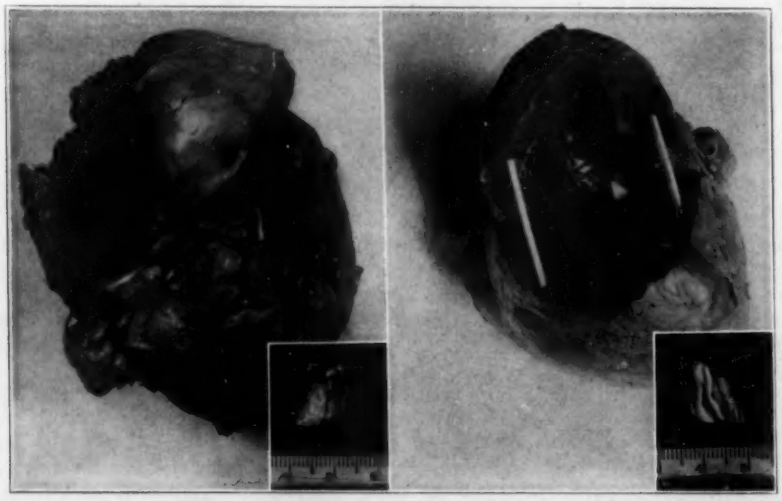

Fig. 6 (case 7).—Auricular and ventricular views of mitral orifice; the insets represent the auricular and ventricular views of the valve segment removed at operation. The operative defect occurs in the aortic cusp without involving the margin of the stenosed valve.

cavity, the rapid formation of which undoubtedly produced tamponade. The mitral ring had been only slightly enlarged by the operation (fig. 5).

Case 7 (Cutler, Levine and Beck[5]).—A young woman, aged 19, did not give any history of rheumatic fever. Shortness of breath on exertion was present for fifteen months. A few months before operation, because of dyspnea, her work as an office clerk had to be discontinued. Palpitation of the heart was present, as were also recurrent attacks of substernal pain. The rhythm of the heart was regular. There was a long, rough crescendo murmur in diastole, a palpable thrill and a systolic blood pressure of 95 mm. of mercury. A diagnosis of mitral stenosis was made. The operation was carried out under ether anesthesia, a midsternal incision being used. The operation of partial valvulectomy was performed, the cardiovalvulotome (fig. 1) being used for the first time. A segment of the mitral valve was excised and removed from the blood stream in the cardiovalvulotome and the incision

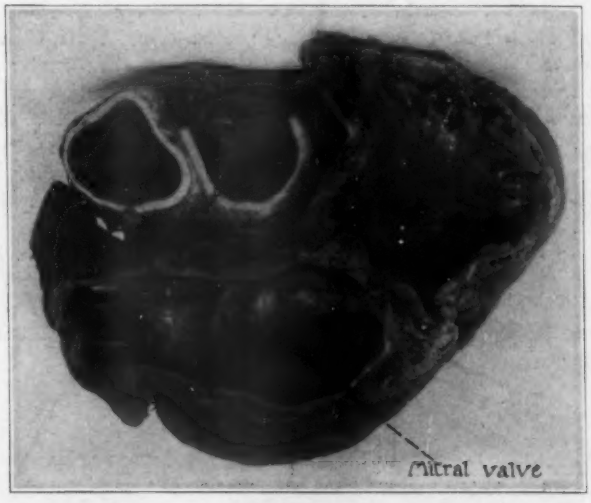

Fig. 7 (case 8).—Auricular view of the mitral valve.

in the ventricle was sutured. The pericardium was not closed completely so that if pericardial fluid should form it might drain into the mediastinum and thus prevent cardiac tamponade. During the first three days after the operation, the condition was apparently satisfactory. The blood pressure was at the preoperative level. The diastolic rumble present before operation had changed to a presystolic murmur. The thrill had disappeared, and a short systolic murmur was heard at the apex. Bilateral pneumonia developed, the heart dilated and death occurred on the sixth day. Autopsy showed bilateral congestion in the lungs with scattered areas of infiltration with leukocytes. An opening about 1 cm. in diameter was present in the aortic cusp (fig. 6) where the cardiovalvulotome had pushed through and excised a fragment from the valve.

Case 8 (Cutler, Levine and Beck[5]).—A woman, aged 21, had rheumatic fever at the age of 14. Dyspnea on exertion, cough and palpitation of the heart had been present for two years. There was no hemoptysis. The left auricle was prominent in the roentgenogram. There was a long rough diastolic murmur and thrill and a

slight systolic murmur. The vital capacity was 80 per cent of normal. The operation was carried out under ether anesthesia and a midsternal incision was used. At operation, two attempts were made to excise a segment from the mitral valve with the cardiovalvulotome inserted into the left ventricle. Each attempt was unsuccessful. The difficulty lay in locating the mitral valve so that the valve could be engaged between the cutting edges of the instrument. Further attempts were not carried out. The pericardium was not completely closed so that cardiac tamponade might be prevented. The patient recovered from the operation. Thirty hours later, signs of pulmonary congestion appeared. The heart dilated, and death occurred on the third day. At necropsy, marked congestion in the lungs was present. The mitral orifice was about 1 cm. in diameter. It had not been enlarged by the operation (fig. 7).

CASE 9 (Souttar[6]).—A young woman, aged 19, presented a history of chorea. Circulatory failure with cyanosis, marked dyspnea and occasional hemoptysis were present for four years. The heart was enlarged and there was some prominence of the anterior thoracic wall. A long diastolic murmur and a soft blowing systolic murmur were present. There was no thrill. The systolic blood pressure was 95 mm. of mercury. A diagnosis of mitral stenosis and insufficiency was made. The operation was carried out under intratracheal ether anesthesia. The thorax was opened by an osteoplastic flap over the second, third and fourth ribs. The lungs partially collapsed as the pleura was opened. The pericardium was opened and the left auricular appendix was drawn into the opening. The base of the appendix was clamped, its tip was incised, the finger was thrust into the auricle, the clamp was removed, and the mitral ring was examined. The mitral orifice readily admitted the finger, thus revealing only a moderate stenosis with little thickening of the valve. The orifice in the valve was dilated by the finger. The finger was withdrawn, and the opening in the auricle was closed with silk. The lung was expanded by increasing the intratracheal pressure; the wound was closed in layers. The recovery from the operation was satisfactory. There seems to have been no change in the physical signs since the operation.

CASE 10 (Pribram[7]).—A woman, aged 28, had rheumatic fever in childhood. For several years dyspnea and palpitation were present, and finally the patient became bedridden, and cyanosis was marked. There was a loud diastolic and presystolic murmur. The pulse rate was irregular; the systolic blood pressure was 90. The diagnosis was mitral stenosis. Under ether anesthesia, the thorax was opened, using the median thoraco-abdominal exposure. The cardiovalvulotome of Beck and Cutler was inserted through the left ventricle; the edge of the mitral valve was engaged between the cutting edges of the instrument, and a segment of the valve was excised and removed. The pericardium was partially closed. The immediate postoperative recovery seemed satisfactory. Auscultation showed marked changes in the diastolic murmur and there was less cyanosis. The temperature rose to 39 C. (102.2 F.) on the second day. Aspiration of the pericardium did not yield fluid. Pneumonia developed, and the patient died on the sixth day. Autopsy showed a freshly recurrent endocarditis with stenosis of the aortic valves. The defect in the mitral valve made at operation was seen (fig. 8). Pulmonary congestion was present.

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6. Souttar, H. S.: The Surgical Treatment of Mitral Stenosis, Brit. M. J. **2**:603, 1925.

7. Pribram, B. O.: Die operative Behandlung der Mitralstenose, Arch. f. klin. Chir. **142**:458, 1926.

Fig. 8 (case 10).—Reproduction of the original illustration from Pribram's article showing the defect created by the cardiovalvulotome.

Fig. 9 (case 11).—Ventricular view of the mitral orifice, showing defect made at operation. The defect was made near the base of the valve.

CASE 11 (Cutler and Beck, first report).—A man, aged 34, had rheumatic fever in childhood. He had attacks of palpitation of the heart for six years; precordial pain, dyspnea on exertion were also present and during the last two years there were frequent attacks of hemoptysis. There was a palpable diastolic thrill. A roentgenogram of the heart showed a prominence in the left auricle. There was a presystolic crescendo murmur. There was no systolic murmur and the heart was irregular. A diagnosis of mitral stenosis was made. Under nitrous oxide-oxygen-ether anesthesia, the median thoraco-abdominal exposure was carried out; the cardiovalvulotome was inserted into the left ventricle. Great difficulty was experienced in locating the orifice of the mitral valve. It was only after considerable effort that a segment of the mitral valve was excised and removed. The pericardium was closed partially to allow drainage. The patient never fully regained consciousness and died sixteen hours after operation with signs of circulatory failure. In this case, the trauma inflicted at operation may have been an important factor in the fatality (fig. 9).

CASE 12 (Cutler and Beck, first report).—A man, aged 47, did not give any history of rheumatic fever. The condition began insidiously, the first symptoms being precordial pain and generalized weakness appearing seven years before. Dyspnea on exertion developed as the next symptom. Three years before, he probably had a pulmonary infection lasting ten days accompanied by fever, cough, sputum, dyspnea and weakness. Following that illness, the patient returned to work, but some weakness persisted. During the last two years, he had to rest about three days every two or three weeks. On one occasion, fluid was aspirated from the chest. He continued his work, which was that of a laborer, with frequent periods of rest until about two months before admission to this hospital. There was a slight degree of cyanosis and an occasional irritating and unproductive cough. The pulse rate was regular. There was some enlargement of the left auricle as shown in the roentgenogram. A diastolic murmur with a presystolic rumble was present, and a diastolic thrill could be felt in the fourth interspace just inside the nipple line. The liver was not enlarged. There was slight edema over the ankles. A diagnosis of mitral stenosis was made.

Operation was carried out under nitrous oxide-oxygen and ether anesthesia. A parasternal incision was made and the costal cartilages of the second, third, fourth and fifth ribs were removed together with a portion of the sternum. The pleura was dissected laterally without opening it and the pericardium was incised. The appendix of the auricle was brought into the wound. An attempt was made to palpate the mitral valve by invaginating the wall of the auricle, but the auricle was so turgid that this was impossible. A clamp was placed on the base of the appendix and the tip of the auricular appendix was incised. The cardiovalvulotome was inserted into the auricle. Some difficulty was experienced in orientating the instrument in relation to the valve. After it was believed that the orifice of the mitral valve had become engaged between the edges of the instrument, the excision of a small piece of tissue was carried out. The instrument was then removed from the heart and the opening in the auricular appendix was closed with a ligature. On making the cut with the cardiovalvulotome, active bleeding occurred so that the pericardium filled with blood. Active bleeding continued for a few minutes, and the heart was finally turned forward so that its appendix protruded into the thoracic wound. The bleeding point was not located. The heart was returned to the pericardial cavity; its stroke was rather feeble and the bleeding had stopped.

The heart soon took on a more vigorous systole, and as the bleeding did not begin again, further manipulation was not carried out to locate and suture the opening which had been accidentally made in it. The systolic blood pressure was 40 mm. of mercury. The pericardium was closed partially, an opening being left to allow the escape of serum or blood to prevent cardiac tamponade. The wound was closed in layers with silk. The patient regained consciousness, but died three hours later from a failing circulation. Postmortem examination was not obtained.

Statistical Table of Operations for Chronic Valvular Disease

Case	Author or Operator	Date	Diagnosis	Method or Instrument	Result
1.	Doyen [1]	1913	Congenital pulmonary stenosis; patent interventricular septum	Tenotome	Died, few hours after operation
2.	Tuffier [2]	1914	Aortic stenosis	Finger dilatation	Recovery, improved
3.	Cutler and Levine: Boston M. & S. J. 188: 1023, 1923	5/20/23	Mitral stenosis	Tenotome	Died, 4 years and 6 months after operation
4.	Allen and Graham [4]	8/ 7/23	Mitral stenosis	Cardioscope	Operative death
5.	Cutler, Levine and Beck [5]	10/ 7/23	Mitral stenosis	Tenotome	Died, 10 hours after operation
6.	Cutler, Levine and Beck [5]	1/12/24	Mitral stenosis	Tenotome	Died, 20 hours after operation
7.	Cutler, Levine and Beck [5]	2/25/24	Mitral stenosis	Cardiovalvulotome	Died, sixth day after operation
8.	Cutler, Levine and Beck [5]	6/11/24	Mitral stenosis	Cardiovalvulotome	Died, third day after operation
9.	Souttar [6]	5/ 6/25	Mitral stenosis and aortic insufficiency	Finger dilatation	Recovery, living and improved
10.	Pribram [7]	11/14/25	Mitral stenosis and aortic vegetative endocarditis	Cardiovalvulotome	Died, sixth day after operation
11.	Cutler and Beck (first report)	12/ 8/26	Mitral stenosis	Cardiovalvulotome	Died, 15 hours after operation
12.	Cutler and Beck (first report)	4/15/28	Mitral stenosis	Cardiovalvulotome	Died, 3 hours after operation
Totals:	12 cases 1 aortic stenosis, acquired 1 pulmonary stenosis, congenital 10 mitral stenosis, acquired		2 finger dilatations 4 tenotome attempts 5 cardiovalvulotome attempts 1 cardioscope attempt		Mortality, 83 per cent

COMMENT

The foregoing summary includes one case of pulmonic stenosis, one case of aortic stenosis and ten cases of mitral stenosis. We have not had any personal experience with pulmonic stenosis and aortic stenosis. It seems that mitral stenosis offers greater promise than do any other of the valvular lesions, and for this reason we shall confine discussion to the cases of mitral stenosis.

Of the ten patients with mitral stenosis who were operated on, only one is living, giving a mortality of 90 per cent. Eight of the ten patients died so soon after operation that the changes brought about in the mechanics of the circulation could not be adequately studied. One

patient lived four and a half years after the operation. It is difficult to say definitely whether in this case the enlargement effected in the mitral valve by the operation was followed by an improvement in the circulation. We believe, however, that there was an improvement in the patient's condition. If it be true that the mechanics of the circulation were improved by reduction of the stenosis, a definite advance in this subject has been brought about. It will require, however, a number of cases in which operation is successful to determine definitely whether an improvement in the circulation can be expected by enlarging the orifice in the stenosed valve. Such physiologic observations have not been produced in animals. Unfortunately, it seems that the basic idea underlying this development will have to be established by attempts on human patients.

If it be taken for granted that the mechanics of the circulation become more compatible with life when the degree of mitral obstruction is decreased, there remain the technical problems of the operation. These problems are: the approach to the valve, the localization of the valve and the question of valvulotomy versus partial valvulectomy. The median sternotomy exposure was used in seven of the ten patients operated on. An excellent exposure of the heart is obtained by this method and it also has the added advantage of not opening the pleura. However, it is an extensive operation in itself, probably too extensive to warrant its use. In one case, an osteoplastic flap was turned back and the pleura opened. In two cases, a less extensive exposure of the heart was obtained by resection of costal cartilages, with or without resection of a portion of the sternum. The type of exposure is determined largely by the method of approach to the valve itself. If the mitral valve is approached through the ventricle, the midline sternotomy or large osteoplastic flap is necessary. If the valve be approached through the auricle, a less extensive exposure by resection of costal cartilages and sternum may be adequate. The ventricular approach to the valve was used in seven cases and the auricular approach in three cases. After having utilized the midline sternotomy and the ventricular approach in six cases, in our seventh case (case 12) we adopted the less radical exposure by resection of costal cartilages and sternum and the approach to the mitral valve through the auricle. It was our hope that the auricle might furnish a more exact approach to the mitral valve. We feel that our failure in the last case cannot be considered too strongly as a contraindication to the auricular approach. The exposure of the auricle is a less extensive operation than that of the ventricle, and it may be possible that instruments with special curves can be made so that the posterior position of the auricle will become less of a handicap in the operation.

Our previously expressed opinion that wide exposure of the heart and approach to the valve through the ventricle is more desirable has

not been altered. We have felt, however, that the great difficulty in locating the stenosed valve from this side, the valve being commonly sucked down into the ventricle and funnel shaped, thus deflecting the tip of the instrument away from the orifice unless it directly engages there at the first attempt, is sufficient to justify further attempts from the auricular side. In approaching from this side, the instrument would constantly be guided into the funnel and toward the valve. In approaching the valve through the auricle, two methods must be considered: (1) a direct attack across the pleural space, so placed that a straight instrument can be pushed from behind and upward, downward and forward, or (2) an attack from in front which may be kept extrapleural, this appearing highly desirable, in which case it will be necessary to have a curved instrument so that though the instrument enters the auricular appendix from in front to behind it would gradually turn downward and finally somewhat forward as it enters the funnel that leads to the valve. Such a curved instrument in our last case might have prevented the unfortunate outcome.

From the foregoing summary of cases, it will be seen that three kinds of procedures were utilized in the attempts to enlarge the stenotic orifice. These methods were finger dilatation, incision of the stenotic valve and excision of a segment of the stenotic valve. We have not had any experience with dilation of the stenosis. We feel, however, that the method may be worthy of trial. A small instrument similar to the Kollmann's dilator of the urologist could be devised, and this instrument could be inserted into the stenotic ring and the latter stretched and dilated. It will be seen that the only two patients of the series living are those on whom dilatation was done. Incision of the stenotic valve was carried out in four cases. From our experience in cases 5 and 6, we felt that the enlargement effected by a simple incision of the stenotic valve was inadequate. We then devised an instrument which could excise a segment from the valve and remove it from the blood stream. This instrument was used in five cases. In each of the four patients that we operated on, some difficulty was experienced in orientation within the heart. We feel at the present time that that is one of the most serious problems in cardiac surgery. The cardioscope devised by Allen and Graham affords a slight degree of visualization of the endocardium at the point of contact with the instrument. The examination that can be carried out with this instrument, however, is so slight that we have not used it in any human cases.

Finally, it may be that we already have the evidence that the success in the finger dilatation method and the success in our first case are due to the fact that only a slight change was made in the size of the orifice of the valve. It may be that the cardiovalvulotome with its actual removal of a piece of valve creates a too sudden change. We know that

all the changes created by nature are slow and gradual; could we return a stenotic valve to the insufficient type by a gradual procedure, we might well achieve success. This question unfortunately cannot be answered until we can experimentally produce stenosis similar to what occurs in man, and then suddenly change this to an insufficiency. We have convinced ourselves that a simple knife cannot enlarge a typically stenosed, thickened and often calcareous valve; we do not know whether the actual removal of a piece of the valve by the powerful cardiovalvulotome is deleterious or not. And we have not yet available evidence as to what excision of a segment of a stenosed valve will result in when the operative procedure is simplified (as performed in our last case), and the postoperative course, therefore, less of a strain on the already lowered vitality.

It may seem that the information obtained from the twelve cases of chronic valvular disease in which operation was performed is so meager that further attempts are not justified. However, in view of the preceding discussion, we feel that a few more attempts are necessary in order to answer certain questions already mentioned. Should it be possible to produce experimental stenoses, these questions could be answered in the laboratory. Unfortunately, our own attempts for seven years along this line have been as unsuccessful as the attempts of other and more experienced investigators.

It is our conclusion that the mortality figures alone should not deter further investigation both clinical and experimental, since they are to be expected in the opening up of any new field for surgical endeavor.

SURGERY OF THE PERICARDIUM AND HEART*

AMBROSE L. LOCKWOOD, M.D., C.M.
TORONTO

The development of surgical procedures on the pericardium and heart to the present status unfolds another wonderful chapter in the romance of the science and art of surgery. It is just thirty-two years since a suture was successfully placed in the pulsating heart. Yet since earliest time, the seriousness of lacerations of the heart has been appreciated and the necessity of pericardial drainage in certain circumstances recognized.

HISTORICAL DATA

Wounds of the Pericardium and Heart.—The evolution of surgical procedures on the pericardium and heart may be studied conveniently in four periods.

First Period: There first existed the legendary period when mysticism prevailed and the heart was looked on as the "fountaine of life, a sacred organ, injury of which was beyond the skill of man to relieve." It began with Hippocrates and ended with Ambroise Paré (1509-1590). It is interesting that Hippocrates did not stress the great danger of wounds of the heart as did later writers. In view of the remarkable appreciation he had of the treatment of intrapleural collections, it is possible that he did not hold the heart in such awe as did the others, and yet in that mystic period he hesitated to record his real feelings in regard to wounds of so sacred an organ.

Ambroise Paré classically described the general opinion in regard to the heart:

> The heart is the chiefe mansion of the Soule, the organe of the vitall faculty, the beginning of life, the fountaine of the vitall spirits, and so consequently the continuall nourisher of the vitall heate, the first living and last dying, which because it must have a naturall motion of itself, was made of dense solide and more compact substance than any other part of the body.

The history of the early wars records instances of spear and lance wounds of the heart and of warriors slowly dying with the weapon penetrating the heart and moving with each beat as "the soul issued in the purple flood" (Homer's "Iliad").

Aristotle (384-322 B. C.) said:

> The heart alone of all viscera cannot withstand serious injury.

Ovid (43 B. C.-17 A. D.) recorded:

> Though Aesculapius himself applies the sacred herbs by no means can he cure a wound of the heart.

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* From the Lockwood Clinic.

Pliny, the elder (A. D. 23-79), wrote:

This is the only one among the viscera that is not affected by maladies, nor is it subject to the ordinary penalties of human life; but when injured, it produces instant death.

Galen (130-200 A. D.), in describing wounds in gladiators, pointed out:

If a ventricle were wounded these gladiators died soon, and especially so if the left ventricle were wounded.

Paré (1509-1590) described vividly the picture of men wounded in the heart. He said:

By these signs you may know that the heart is wounded; if a great quantity of blood gush out, if a trembling possess all the members of the body, if the pulse be little and faint, if the colour become pale, if a cold sweate and frequent sowning assayle him, and the extreme parts become cold, then death's at hand.

Fallopius (1523-1562) asserted:

Wounds of the heart are always followed by sudden death. When wounded it cannot heal, being too firm, always in motion, and of an inflammatory heat.

In recording the instance of a patient with a needle wound of the heart, in whom death did not occur till the sixth day, Zacchias (1584-1659) said:

The noblest organ of the body cannot survive a solution of its continuity and this is the general medical opinion.

Boerhaave (1668-1738) stated:

All wounds of the heart deep enough to penetrate into either of its ventricles are mortal.

Paul Barbette (1689), Amsterdam, wrote as follows:

Wounds of the heart are always mortal and those that penetrate into the left ventricle kill suddenly. Those so wounded seldom live above 6, 12 or 20 hours,—although there are examples to the contrary. A wound in the right ventricle permits the patient to live longer, and one which terminates in the substance of the heart grants yet longer time.

Second Period: The second period was characterized by an era of experimentation and independent thought in medicine, as in the other sciences and classical learning generally. It dated from Paré (1590) to George Fischer (1868). It was no longer heresy to question or depart from the teaching of the ancients. Physiology and pathology were studied. Experimental work was carried on, and treatment developed. It was the period of Riolan, Harvey, Morgagni and Larrey and ended with Fischer's wonderful monograph, containing all data and knowledge

obtainable up to that time. Fischer's treatise was the greatest factor up to the close of that period in stimulating members of the medical profession to consider the possibilities of cardiac surgery.

Hollerius (1498-1562) was the first to advance boldly the theory that wounds of the heart are not necessarily fatal and can heal. In 1749, Sénac pointed out that wounds of the heart heal and are not always fatal. In 1761, Morgagni was the first to stress the danger of compression of the heart from hemorrhage into the pericardial sac, which Rose, in 1884, termed "hertz tamponade."

Treatment during this period consisted of rest, the use of leeches, venesection, cupping and the passage of a catheter or sound into the wound to allow the fluid to escape from the pericardial cavity, and thus relieve pressure.

In 1829, Larrey reported a successful drainage of the pericardium. He inserted a catheter through the stab wound and drained off three beakers of wine-colored fluid. Dupuytren (1834) advocated venesection almost to the point of exsanguination. Jobert, in 1839, stressed the danger of increased intrapericardiac pressure, and pointed out that death occurred because the blood shed into the pericardial cavity did not escape through the wound in the pericardium and exerted such pressure that the heart could not function.

In 1850, Purple collected forty-two cases of wounds of the pericardium and heart. Eleven of these were reported in America.

Fischer's classic article appeared in 1868. He recorded 452 cases, 90 per cent of which were fatal.

Third Period: The third period began with Fischer, in 1868, and ended with Rehn, in 1896.

With the advent of antiseptics and asepsis in the second half of the nineteenth century, experimental surgical procedures were undertaken on animals. The tolerance of the heart to surgical manipulation and suture was established. In December, 1881, Roberts of Philadelphia first suggested that it would be rational to attempt suture of wounds of the heart. In 1882, Block first sutured lacerations made in the hearts of rabbits.

In 1887, Reed, of Cincinnati, successfully sutured a wound of the pericardium. In 1891, Dalton, of St. Louis, also performed a successful suture of the pericardium. Rosenthal, Salomoni, Del Vecchio, Bonomi and Martinotti (1888) contributed to the advance in this section of surgery.

In 1883, Billroth stated:

> Let no man who hopes to retain the respect of his medical brethren dare to operate on the human heart.

In 1888, Riedinger wrote:

> The suggestion to suture a wound of the heart, although made in all seriousness, scarcely deserves notice.

Nicholas Senn, one of the most daring and astute surgeons of his time, pointed out that "surgical interference with the heart was impracticable."

In 1896, Stephen Paget stated that:

> Surgery of the heart has probably reached the limits set by nature to all surgery; no new method and no new discovery can overcome the natural difficulties that attend a wound of the heart.

Little encouragement was given to the work by the great masters of surgery of the time, until 1895, when Del Vecchio at the meeting of the Eleventh International Medical Congress at Rome, demonstrated healed wounds of the heart in dogs following suture. Within a year, three attempts were made to suture wounds of the heart in human beings. The first attempt was made by Cappelen, of Christiania, on Sept. 4, 1895; the second by Farina, of Rome, in March, 1896; both attempts were followed by death. The third and first successful attempt was made by Rehn, of Frankfurt, on Sept. 9, 1896.

Fourth Period: The final period, ushered in by Rehn in 1896, was characterized by a widespread interest and appreciation of the possibilities of cardiac surgical procedures. Within ten years of the first successful case of suture by Rehn, in 1896, he was able to collect 124 recorded cases, with recovery in 40 per cent. In 1906, Borchardt reported seventy-eight cases in which operation was performed with thirty-nine deaths, twenty-one of which were due to infection. Up to Jan. 1, 1906, Salomoni compiled statistics of 160 cases with recovery in 62 per cent. In 1909, Vaughan reported 150 cases with recovery in 35 per cent. Fourteen of these were reported from America, with six recoveries. Forty-four of the patients who died lived at least twenty hours after operation, and died mainly of sepsis.

In 1909, Peck collected and exhaustively tabulated 160 cases in which operation was performed by 121 different operators, with recovery in 36.2 per cent, no surgeon having operated in more than three cases.

In 1912, Pool collected seventy-nine cases reported in 1909, 1910 and 1911, in addition to some earlier cases not previously reported. With Peck's list, these cases made a total of 236. From 1912 to August, 1914 (the outbreak of the Great War), at least seventy-five operations on the heart were reported. Approximately sixty cases, according to Ballance, were reported during the war. Personally, I am positive that operation was performed in a great many more cases and never reported, because of pressure of work, loss of statistics, deaths among surgeons

in advanced operating centers, and such contingencies. In the fifty-eight case reports collected by Ballance from war records, forty-four patients recovered and fourteen died, making recovery in 75.8 per cent of the cases.

In 1920, Constantini collected 287 cases of injuries to the heart with recovery in 141, or 49.12 per cent, following operation.

In the same year, Tuffier reported 305 collected cases with recovery in 50.4 per cent.

In 1921, Freese reported a successful suture of a stab wound through both ventricles of the heart, with recovery. The patient was operated on approximately one hour after being wounded.

Smith collected fifty-eight cases from 1912 to 1923, with recovery in 66.6 per cent.

In 1923, Klose compiled records of fifty-seven cases of gunshot wounds with recovery in 68.6 per cent of thirty-two patients operated on, and 62.8 per cent in twenty-four patients treated conservatively. He reported a later successful operation for gunshot wound with recovery in 1926.

In 1924, J. J. Dshanelidze reported 535 cases of wounds occurring from 1896 to 1921. Of these, 403 were cases of stab wounds and 132 were caused by missiles; 44 per cent of the persons recovered. Of 113 cases traced from two months to eighteen years, imperfect cardiac function existed in only 3.5 per cent due to adhesions, weakening of the scar and aneurysm.

In 1926, Lenormant reported a successful suture of a stab wound of the heart. He had three such cases with recovery in one.

In the same year, Bufalini reported two cases of stab wounds with recovery following operation.

The staff of the Lockwood Clinic has compiled fifty-one cases with recovery in 78.4 per cent. So far as I know, these cases have not heretofore been collected.

COMPARISON OF TYPES OF INJURY

There is no doubt that gunshot wounds of the heart are more dangerous than other injuries. In Rehn's list, compiled in April, 1907, 109 were cases of stab wounds and only fifteen, or 12 per cent, gunshot wounds. Of 128 cases reported by Lenormant in September, 1906, only sixteen were caused by gunshot.

Of the 535 cases compiled by Dshanelidze, in 1924, only 132 were due to projectiles. Loison's statement that at least one third of the persons with gunshot wounds of the heart die immediately or within the first hour after the injury is borne out by the experience of many surgeons in dealing with large numbers of seriously wounded men during the Great War. While, undoubtedly, many gunshot wounds of

the pericardium and heart must have been observed in casualty clearing stations and advanced operating centers after early surgical intervention in thoracic lesions had been adopted, it is significant how few cases were reported as having been observed at either operation or autopsy. Henry and Elliott, reporting the autopsy observations in 100 cases of wounds of the thorax, found only one cardiac lesion, and it was not the immediate cause of death. Dixon and McEwan observed one case of wound of the heart in 123 gunshot wounds of the chest. Among 199 patients with wounds of the chest, whose cases were reported by Roberts and Craig, one of the thirty-three who died had laceration of the pericardium. Lockwood and Nixon found only one wound in the heart in forty cases of fatal abdominothoracic injuries which came to autopsy consecutively in the autumn of 1916. We observed, however, many patients with all the clinical evidence of hemopneumopericardium due, at least, to trauma to the heart and pericardium.

During the war it was not uncommon, when doing a thoracotomy for a gunshot wound of the chest, to observe ecchymosis of the pericardium. It was seldom that an actual rent of the pericardium was found, although in retrospect one is convinced that concomitant injuries of the pericardium and heart were present in some of the cases which were postoperatively fatal, but because of constant pressure of new cases and the shortage of skilled men, autopsies were possible in only a limited number. It is significant, however, and confirmatory of the seriousness of wounds of the heart and pericardium during the war, that in the hundreds of gunshot wounds of the chest that were cared for, I did not observe one case of the so-called "hertz tamponade," nor did a man with a bayonet wound of the heart or pericardium survive long enough to reach an advanced operating center or casualty clearing station. In only three cases were surgical procedures undertaken for removal of a missile from the heart, and in two cases for suture of the pericardium per se. These will be reported later. Six cases were observed in which missiles were found either within the heart cavities or in the circulation, which previously had been in the heart.

In 1915, Sir Henry Gray operated on a man who had been wounded fifteen days previously. He had had little disturbance, apart from running a fever from 100 to 101 F., until the fourteenth day when he developed a sharp pain in the left leg and the pulse rate became irregular (from 65 to 95). Roentgen examination revealed a bullet in the heart. The next morning, with the man under local anesthesia, the sixth costal cartilage was served, the sternum sectioned transversely and the heart thus exposed. The bullet was located in the posterior wall of the right ventricle, free in the cavity. With some difficulty, it was pinched between the thumb and finger, two mattress sutures placed, an incision made onto the bullet, which was extracted, and the sutures tied. A

running suture was then added, and the pericardium closed. Four hours afterward, the respirations rose to forty-eight, and remained so till death ensued on the fifth day. Autopsy revealed clots entangled in the cordae tendinae and a long narrow clot in the pulmonary artery. The entrance wound of the bullet was not discovered.

John Fraser (Edinburg), working in No. 6 Casualty Clearing Station, observed two deaths from herz tamponade six and eight hours, respectively, after admission. In a third case, he performed an operation, evacuated a large amount of blood from the pericardium and with two sutures closed a laceration in the anterior surface of the right ventricle. The patient recovered.

Major H. H. Sampson reported a successful suture of a laceration of the left ventricle with an extensive tear of the pericardium, and a wound of the left lung with a large hemothorax. Ten days later, the patient was evacuated in good condition. Such cases reflect the great advance of thoracic surgery in 1916, 1917 and 1918.

Sir Berkeley Moynihan recorded a case in which he removed a shrapnel bullet from the wall of the left ventricle twelve and a half months after injury. This case illustrates well the contention of Sir Berkeley that early operation in such cases would avoid infection and consequent pericarditis which have caused so many of the deaths or months of distress to patients who ultimately required surgical treatment.

Briggs, working at a casualty clearing station, recorded the removal of a bullet from the wall of the ventricle. Death ensued eight hours later, due to septic pericarditis.

G. Gillen, in No. 3 Canadian Stationary Hospital, sutured the pericardium in two cases of gunshot wounds; one of the patients recovered.

In 1918, Colonel Sam Streight and Major H. G. Wood operated on a wounded boy, 19 years of age. He had been comfortable for twenty-four hours after the wound was received, when he developed intense spasmodic precordial pain. Donaldson, one of the most expert of the R. A. M. C. radiographers, localized the missile in the right ventricle. At operation, the missile could not be located. The patient died from four to five days later, and autopsy revealed a flat piece of shrapnel (tin), half the size of the little finger nail, caught in the cordae tendinae of the right ventricle. The missile had entered the lower edge of the liver, passed through the anterior wall of the hepatic vein and was carried to the auricle and thence to the ventricle. Death was due to pericarditis.

My first case was that of Private S., aged 26. There was a wide entrance wound just to the left of the sternum in the fifth interspace. A piece of shrapnel, 1 by 2 cm., was lodged in the wound beneath the sternum, and had chipped the

edge of it. The patient had precordial pain, tachycardia and was febrile. Ten hours after being wounded, the wound was widely excised; the incision extended from the third cartilage above to the sixth costal cartilage below. The fourth, fifth and sixth cartilages were severed. The internal mammary artery was retracted, and the triangularis sterni freed. The osteoplastic flap was retracted laterally, freely exposing the pericardium. A laceration 3 cm. long was found in the pericardium with the missile projecting into it. The missile was removed. The edge of the rent in the pericardium was excised and the pericardium widely incised. There was some blood-stained serous fluid in the sac and a little blood clot. A small area of ecchymosis was found over the right ventricle, without actual laceration. The sac was carefully sponged out with absorbent cotton and closed without drainage. A suture was carried from the inner edge of the sutured pericardium to the surface. The flap was replaced, and the wound closed without drainage. The wound healed by first intention. On the seventh day, the suture extending to the pericardium was withdrawn. The patient had an uninterrupted convalescence. He was kept in the unit on light duty for six weeks, evacuated to England, and three months later returned to his battalion.

My second case was that of Sergeant F., aged 37, who had an extensive shrapnel wound of the left side of the chest in the seventh and eighth space in the mid-axillary line with laceration of the lower lobe of the lung. The missile measured 3.5 by 2 by 1 cm., and was embedded in the mediastinum posteriorly. He had dyspnea, precordial pain referred through to the back, a constant hacking cough with hemoptysis, and all the signs of a large hemothorax. He had a tumultuous and irregular heart beat with the churning sound which we (Lockwood and Nixon) had come to recognize as due to a hemopneumopericardium. We had determined long before this, however, that such phenomena could occur from trauma to the heart resulting from a blow by a large missile which might rebound and, on roentgen examination and at operation or autopsy, be found lying at some distance from the heart or directly posteriorly, so that with each beat of the heart its posterior aspect touched the missile. Autopsy had revealed two such cases with severe bruising of the heart, but without extensive hemopericardium, laceration of the pericardium or hemorrhage into the cavities of the heart. In this case, eight hours after the patient was wounded, the chest was widely opened. The missile had torn the pericardium laterally and posteriorly just behind the apex for approximately 4 cm. There was no evidence of injury to the heart and no fresh hemorrhage from within the sac. The pericardium was sutured without drainage. The missile was removed from the posterior mediastinum where it was found embedded. After clearing out the hemothorax completely and excising the wound in the lung, the chest was closed without drainage. The patient made a good recovery.

In my third case, Private B., previously reported, aged 19, received a wound from a missile which traversed the right side of the chest (hemothorax) and mediastinum, and lodged in the apex of the left ventricle of the heart.

He was admitted to No. 36 Casualty Clearing Station on Aug. 7, 1917. The entry wound was in the right posterior axillary line. There was closed hemothorax (right). On roentgen examination the missile was located on the left side, presumably behind the pericardium. Cardiac irregularity was not noted, nor were there any signs of air or fluid in the pericardium. Hemothorax was aspirated on the fifth day (35 ounces, noninfected).

Fourteen days after the wound was received, on account of continued pyrexia and the missile being retained, operation was performed. Omnopon-scopolamine, paravertebral and local anesthesia (no gas and oxygen) were used. The chest was

opened by parasternal incision through the fourth, fifth and sixth cartilages on the left side. The missile was searched for behind the pericardium, but was not found. There was hemothorax on the left side. On palpating the pericardium and heart, the missile was felt in the heart muscle. The pericardium was widely opened, and a piece of shrapnel 1 by 1 cm. was found lying superficially in the muscle on the posterior aspect of the left ventricle—medially and about 2 inches (5 cm.) from the apex. Fluid was not found in the pericardium; there was a thin, non-adhesive, serofibrinous film covering the visceral and parietal pericardium. Unfortunately, owing to the difficulty of accurate localization of the missile in the heart by roentgen examination, the pleural cavity had been unnecessarily searched. Even the gentlest attempts to grasp the heart laterally, or to raise it by a stay suture in the apex, excited fibrillation or caused it to stop beating. As fourteen days had elapsed since the wound was received, and on palpation there was no inflammatory process round the missile, it was judged wise to leave it in situ. The pericardium and chest were closed without drainage. When the heart and pericardium were handled, the patient did not complain of pain or distress, either cardiac or respiratory, although only local anesthesia was used. It was not possible to expose the area over the missile so that it could be easily and safely incised and sutured.

The wound healed by first intention. The patient made an uninterrupted recovery. Cardiac irregularity was not observed after operation. He was out of bed three weeks before evacuation, doing light duty in the ward. He was evacuated on Nov. 4, 1917.

Had the missile been accurately localized so that it was necessary to open only the pericardium it is possible that the heart would have been more resistant to elevation and rotation. This patient was followed for five years, and was still in good health.

I was relieved to find the report of a similar case by Jenckel. In his case, the bullet was embedded in the posterior wall of the right ventricle. Operation six months after injury had to be abandoned, because the heart could not be brought forward enough to expose the posterior surface of the ventricle. Six months later, Jenckel had reoperated on the patient and succeeded in removing the bullet; the patient recovered.

Sir Henry W. Gray was unsuccessful in a case at No. 3 Canadian Stationary Hospital. The missile was located in the right ventricle by roentgen examination, but at operation it could not be palpated nor located with a needle. The patient died four days later, and the metal, a thin piece of shell casing, was found at autopsy.

MIGRATION OF FOREIGN BODIES

The migration of foreign bodies, such as bullets, needles and shrapnel, to and from the heart, is most interesting. In the treatment of gunshot wounds, one is soon impressed with the fact that regardless of the site of entry, the missle, particularly if it is a rifle or shrapnel bullet, may travel almost anywhere within the body and lodge in the least suspected area. Many interesting cases of migration of missiles in the blood stream have been reported.

Hamilton, Fayrer, Simmonds, Randall, Fischer, Loison, Salomoni, Socin, Riedinger, Bergmann, Elton and many others have reported experiences with such cases. In 1903, H. Schloffer presented a most convincing paper on the tolerance of the heart to foreign bodies.

Salomoni (1901) reported ninety-seven cases of foreign bodies in the heart, six of which entered by the esophageal route; all of the cases were fatal. Fifty-one of the ninety-one cases were fatal and recovery occurred in forty.

The Vienna correspondent of *Lancet* reported the case of a man who shot himself in the left side of the chest. Five hours after the bullet was found to have penetrated the heart, a swelling of the right leg developed, extending to the knee. Roentgen examination revealed the bullet lodged in the right femoral artery, opposite the head of the femur. The artery was incised, the bullet removed and the vessel sutured. Later, the leg had to be amputated, but the patient recovered. At that time only seventeen cases of migration of a bullet to the peripheral vessels could be collected. Thirteen of these cases were diagnosed during life; operation was performed in only three, with recovery in all. The correspondent's case was included in one of the three. Many interesting cases were reported from the Great War. Burckardt found a projectile in the pulmonary artery.

Ascoli and Mansoni cited a case in which a ball entered the left iliac vein and was carried to the right auricle. The patient was not operated on, and had little discomfort from the missile retained in the heart.

Perdoux successfully removed a bullet from the right iliac artery that had entered through the left ventricle.

Viscontini removed a ball from the right ventricle that had entered through the pulmonary artery.

In 1916, Dencke successfully removed a bullet from the right axillary artery that had been carried in the blood stream from the left ventricle. In a second case, the bullet was carried into the right subclavian artery and removed through incisions of the vessel, but the patient died later of sepsis and secondary hemorrhage.

Simmonds reported a case in which a pistol ball that had entered the vena cava inferior was carried to the right ventricle, and thence to one of the iliac veins where it was removed.

Duval reported twelve cases with deaths in nine in which the missile entered the heart through the blood stream from peripheral or distant vessels.

In one of the cases seen overseas the patient was admitted approximately six hours after being wounded. He was in extremis. There was a small entrance wound on the right side of the abdomen just below the tip of the ninth rib. It was not thought that he had any great amount of intra-abdominal hemorrhage. The pulse rate was irregular and fast,

from 140 to 160; he complained of precordial distress, and the heart beat was tumultuous. Roentgen examination was not available. He died soon after admission. Autopsy revealed that the missile, a small piece of shrapnel, had entered the vena cava inferior, and it was found lodged in the left ventricle. There was practically no bleeding from the small rent in the vena cava. The cause of death was not clear. There was no pulmonary embolism.

In two cases a rifle bullet that had entered the chest penetrated the heart; in one case it lodged in the right femoral artery and in the other in the right external iliac. Both patients were in extremis; they were admitted to the casualty clearing station approximately from eight to ten hours after being wounded, and died shortly after admission. In one, the bullet had entered through the pulmonary vein; in the other, the entrance wound to the heart could not be found.

A fourth patient was admitted approximately from six to eight hours after being wounded, with a small penetrating wound in the third interspace on the left side just at the outer border of the sternum. His condition was good. Eight hours after admission, he was sent to the x-ray department for localization of the missile. About 6 p. m., a small piece of shrapnel was located in the left ventricle, moving tumultously about with each beat of the heart. About 7 p. m., the patient began to complain of intense precordial pain, the pulse rate became irregular and fast from 160 to 180. The temperature, which had been 99.2 F., suddenly rose to 106 F. Surgical intervention was considered urgent. At 8:15 p. m., the patient died suddenly of what appeared to be pulmonary embolism. Autopsy revealed the missile in the right popliteal space. The pericardium was apparently intact. There was no blood within the pericardial cavity. There was a little blood clot in the left ventricle and some bruising of the columnae carneae, but no positive entrance wound could be demonstrated in the heart itself.

Bland-Sutton reported a most extraordinary case of a boy with a piece of wood in the ventricle that had entered through the pulmonary vein. The patient lived for thirty-seven days.

LaRoque (Richmond, 1926) recorded the case of a man shot on May 6, in the back on the left side just to the inner aspect of the vertebral border of the scapula. He ran some distance, when the left leg gave way. Roentgen examination the next day revealed a fragment of bullet in the left side of the chest and another fragment 2 inches below Poupart's ligament in the region of the femoral artery. On May 18, the left side of the chest was opened and considerably clotted blood was removed. On July 25, the femoral artery and vein were exposed and 2 inches of the artery enclosing the bullet and the vein were excised. Collateral circulation was well established, and there was no further trouble with the leg.

The heart or aorta may be perforated with a bullet without causing death. A missile may enter the hepatic vein or the vena cava, produce practically no local haemorrhage and be carried to the right ventricle. The pulmonary vein may be entered and the missile be carried to the left ventricle. A bullet may be ejected from the right ventricle into the aorta. A bullet may remain in a ventricle and be swirled about by the blood current or be ejected from the right ventricle into the upper main branch of the right pulmonary artery as in Fullerton's case. (Ballance.)

From the right ventricle an embolus or foreign body may obstruct one of the large pulmonary vessels, where it is not easy to remove it, and where it is not well borne, and thus will be a grave menace. Hence, a prompt operation for removal of a missile from the right ventricle is indicated. On the other hand, the missile may pass from the left ventricle to the subclavian, axillary, iliac vessels or lodge in a peripheral vessel, from any of which it can be removed with safety. It is probably better not to be too hasty in undertaking cardiotomy for removal of a missile loose in the cavity of the left ventricle. It is much less risky to remove it as an embolus in a distant vessel.

REMOVAL OF FOREIGN BODIES FROM THE HEART

Rehn emphasized the necessity for immediate removal of needles, but advised that broken pieces of knives, daggers, etc., should not be withdrawn till all preparations are made for immediate cardiorrhaphy, if required.

Zesas advised removal of all foreign bodies in the heart. In 118 cases, ninety-six entered through the chest, twelve from the esophagus, four by the blood stream and one from the bronchial tubes. In fifty-four cases there were needles in the heart and bullets in thirty-eight. Nails, splinters, thorns, iron pegs, pieces of bone and hairpins have been found. Hoch found a nail embedded in the heart that had caused no apparent trouble.

Paul Delbet reported 115 cases during the Great War; thirty-seven were cases of stab wounds; eight, puncture wounds or crushing wounds, and seventy were caused by projectiles.

Schneider collected thirty-six cases in which there were projectiles in the wall or cavities of the heart.

Missiles may pierce the pericardium, injure the heart, rebound and remain fixed in the lung. They may lodge in the mediastinum, the pericardium itself, the sac of the pericardium, the wall of the heart, or enter a compartment of the heart and remain there or be carried peripherally in the blood stream. They may pass through the heart and remain embedded elsewhere or pass out of the body altogether. They may cause acute distress that subsides or rapidly becomes more severe, or they may cause instant death. They may remain inert or be passed

out of the heart into the blood stream. It is not easy to be sure how extensive the injury is. In those cases in which death is not immediate, pressure from hemopericardium is the gravest complication. The auricles are compressed, venous obstruction occurs, precordial distress, cyanosis and dyspnea increase, the blood pressure falls, the respirations are shallow, the pulse rate is irregular, rapid and thready and of low tension, and immediate relief from pressure by pericardiotomy is necessary.

In 1917, Kuno demonstrated experimentally that when the pressure in the pericardium attained the height of the venous pressure, the heart ceased to function.

Bleeding into the pericardium is usually limited to 250 Gm. Deroide reported a case of hemopericardium without a lesion of the pericardium itself, with 500 Gm. of blood in the pericardial sac. A complicating hemothorax may contain from 1,000 to 1,800 Gm.

If hertz tamponade does not occur, the blood may escape into the pleural cavity and the patient present the picture of hemorrhage with increasing respiratory distress from pressure of the hemothorax. The "hemopleuralpericardial knock" described by Lockwood and Nixon, Rees and Hughes and Maynard Smith is usually present. This is the bruit de Moulin observed and reported by Brauer in thirty-eight cases in 1844. Tuffier speaks of it as a "Souffle Systolique." Surgical intervention is urgently necessary to control the hemorrhage and clear out the hemothorax.

There may not be much hemorrhage and little cardiac distress, but infection may be carried in and a rapid or slowly developing purulent pericarditis may develop. Gas gangrene of the pericardium and of the heart has occurred.

It is possible to have a serious wound of the heart without an injury to the pericardium. These contusion wounds of the heart are due to a sudden blow over that organ caused by a fall, a kick from a horse, etc., and occasionally to a sharp-edged missile, carried from a distant vein into a cavity of the heart and lacerating the heart muscles or valves as it is swirled about. In the great majority of cases, however, the pericardium is involved. In a high percentage, the pleura as well is lacerated. Hesse reported this condition in 80 per cent of his cases. Goebell, in collected cases, found it occurring in 47 per cent; Loison, 45 and Guibal, in 89 per cent.

Injury to the bundle of His, as a rule, is rapidly fatal. Laceration of the coronary vessels is usually fatal. Wounds of the coronary arteries produced by traumatism or punctures in the course of operations have been reported. Pagentescher, Bradbury, Wilms and Gregoire have ligated the postcoronary artery. Hallopeau has ligated the anterior coronary, and Ombrédanne and Rouvillois have ligated a branch of the left coronary artery without any trouble. Wounds of the auricle are

more dangerous than wounds of the ventricles, and wounds of the right ventricle are more serious than those of the left. Wounds of the right ventricle tend to bleed continuously, while lacerations of the left ventricle bleed in spurts, intermittently. Wounds of the ventricles have undoubtedly healed spontaneously, and some have ruptured again later.

In 1868, Fischer declared that cure occurs spontaneously in from 9 to 10 per cent. In 1913, Leotta stated that this percentage was from 8 to 9. Aneurysms in spontaneously healed wounds have developed, and have later ruptured into the pericardium. Loison reported nine deaths caused by fatal hemorrhage from aneurysms in cases in which the wound was left to heal spontaneously. Wounds of the thin-walled auricles do not tend to close.

Curious deaths occur in heart wounds without any clear cause.

Marais reported the instance of a patient who had a superficial wound of the chest directly over the right anterior surface of the right ventricle. The patient lived one day. Autopsy revealed three fissures opening into the ventricle. There was not enough hemopericardium to affect the heart by pressure.

Geibach reported a case in which a needle had perforated the pericadium and wounded the heart. Six weeks later, death occurred suddenly as the patient raised the left arm. There was no blood in the pericardium.

Gallard reported a case in which a patient, wounded with a needle, returned to work on the fourth day, and died suddenly on the thirty-first day. Autopsy revealed the pericardium full of blood. Kraukoff and Messhi have each reported a similar death on the thirty-third and twenty-first days.

Nast-Kolb reported the case of a patient with a shrapnel ball impinged in the heart muscle; in removing the ball through the track of it, a fatal hemorrhage ensued.

Ewald reported a case in which several secondary hemorrhages finally caused death.

Fischer reported a secondary hemorrhage on the ninth day.

The value of early surgical intervention in cases of wounds of the heart is fully established by a study of recorded cases. It must be borne in mind, however, that results in the large series of collected cases appear more favorable than is actually the case. This is due to two reasons: (1) favorable results are invariably more freely recorded than failures, and (2) on a close study of the actual injuries of the heart there is a great difference in severity of the lesion, depending on the portion of the heart involved, and whether or not a cavity of the heart has been entered.

In recording future cases, it would be extremely valuable if a sharp distinction were made between cases in which a cavity of the heart is

opened, and those in which the wall only has been involved without entering a cavity.

RADIOGRAPHY

The value of thorough radiologic examination cannot be overestimated. A skilled radiographer can localize a missile with the greatest accuracy. Gas or air localized in the pericardial sac may be the first guide to a serious lesion. The presence of hemothorax, hematoma, pneumothorax, distention of the pericardium due to serous effusion, pus or blood, partial collapse of a lung and the position of the heart, mediastinum and diaphragm can be determined.

The surgeon, physician and radiographer should be closely associated in the care of these patients.

TABLE 1.—*Statistical Results of Fischer, Borcardt, Rhen, Vaughan, Peck and Pool (up to 1912)*

Punctured wounds	44	Gunshot wounds	72
Incised wounds	260	Crush of chest	76
Total	452 cases		
Right ventricle	123	Interventricular septum	7
Left ventricle	101	Entire heart	16
Both ventricles	26	Right side of heart	4
Right auricle	28	Left side of heart	5
Left auricle	13	Coronary artery	2
Apex	16	Unspecified	57
Base	3	Pericardium	51
Total	452 cases		

Foreign bodies in heart in forty-seven cases: needles, 18; bullets and shot, 13; miscellaneous, 16.

Recoveries		Deaths	
Heart wounds	50	Within a few minutes	104
Pericardial wounds	22	Later	219
		Time not stated	57
Total	72	Total deaths	380

OPERATIONS

The operations now practiced on the heart are:

Sympathectomy for angina pectoris.
Section of the valves in mitral stenosis.
For cardiac stimulation—massage.
Paracentesis of the right auricle and ventricle.
For pulmonary embolism.
Paracentesis, pericardotomy.
Cardiolysis (for chronic adherent pericarditis—Pick's disease).
Cardiorrhaphy—operation for injury.

Sympathectomy for Angina Pectoris.—The experiences of the last decade in regard to sympathectomy for relief from distress in angina pectoris are hopeful. Patients have been definitely benefited. Careful selection of cases for operation and close study with observation of the

type and extent of the sympathectomy promise to establish a routine operative procedure in the near future that will relieve distress from this grave disease. Close cooperation between the surgeon, cardiologist and physiologist is essential. Care should be taken that the procedure is not too widely adopted until a closer study of patients operated on by those specially interested, determines the type of case to select and the extent of the sympathectomy to be performed.

Treatment of Mitral Stenosis (Section of the Valves).—The principle of overcoming mechanical obstruction of the mitral valves by section may yet be solved. In 1902, Samways and Brunton first proposed such a possibility. Klebs, in 1875, Cushing, in 1907, Schepelmann, in 1912, and Tuffier and Carrel, in 1913 and 1914, experimentally demonstrated the feasibility of such a procedure. Elliott Cutler and his associates deserve the entire credit of having established the procedure as possible in human beings. After years of most thorough experimentation on animals, Cutler has operated in seven cases, all of which were desperate cases of mitral stenosis. The condition of one patient was improved four years after operation but death occurred, pneumonia following violent exertion. The other patients lived from six hours to seven days. Allen and Graham operated on one patient, approaching the mitral valve through the auricle rather than the ventricle. The patient did not survive.

The only patients that have survived interference with the mitral valve, apart from Cutler's patient, are two in whom the valve was dilated by the fingers. One case was reported by Tuffier and Carrel and one by Souttar.

The early postoperative death in six of Cutler's cases suggests that perhaps the circulation is altered too suddenly by cutting or incising the valves, and that dilatation may be sufficient.

The safety and ultimate value of section of the valves could much more readily be determined, if it were possible to produce typical mitral stenosis, experimentally in dogs. So far experimenters have not succeeded in producing such mitral stenosis in animals. Allen and Graham have contributed to the work by the development of their most ingenious cardioscope, thereby permitting actual section of the valve under direct vision. Jarotzky suggested making an artificial communication between the auricles. It is known that mitral stenosis complicated with an open foramen ovale has a comparatively good prognosis. The operation could be performed by the simple way of introduction of the instrument through the jugular vein.

With further development of Cutler's cardiovalvulotome and the cardioscope, and additional experience as to the proper approach and extent of the section, mitral stenosis, that scourge of heart disease, seems about to enter the realm of surgery, with great promise of dis-

tress being relieved and of life being lengthened in those victims of an otherwise hopeless malady.

Exposure of the Heart to Permit of Cardiac Massage.—Several methods have been advised to expose the heart so that it may be rhythmically massaged to restore movement, when it has become suddenly arrested. One method is by indirect massage by pressure over the precordial area, and the other by direct massage by the thoracic, subdiaphragmatic or the transdiaphragmatic route.

Tuffier, in 1897, was the first surgeon who deliberately opened the thorax to permit of cardiac massage. He made an incision in the fourth interspace on the left side, close to the sternum; he introduced the fingers through the opening and rhythmically massaged the ventricles. The heart had ceased to beat during an operation under chloroform anesthesia. Ten minutes elapsed before the heart began to function. The patient recovered. Tuffier and Hallium reported the operation the next year.

Jeanbrau restored cardiac function thirty minutes after the heart had ceased to beat, and Lenormant restored contractions after one hour, but the patient did not return to consciousness, and died.

The transdiaphragmatic route is indicated in a small percentage of patients in whom the diaphragm is so firmly contracted that it is not possible to grasp the ventricles between the thumb and the second and third fingers (Green, Sencert and Gross and Mauclaire).

The subdiaphragmatic route allows rapid access to the heart, and in the great majority of patients the heart can be readily grasped in the hand.

Tomaseli proposed the subdiaphragmatic approach, but without opening the peritoneum. Undue delay, however, in displacing the peritoneum would not be justified if the heart had been arrested some minutes.

Wrédé demonstrated the effectiveness of cardiac massage. After death he injected a little colored fluid into the jugular vein, massaged the heart and found the fluid in the distal arteries and the portal vein. In addition, he observed a reflex contraction of the ventricles.

The intravenous injection of saline has been shown definitely to set up contraction of the ventricles. Direct injection of epinephrine into the heart muscles will excite contractions. In only one case did the heart respond after the second injection. All these accessory measures should be adopted at once in cases of cardiac failure. In 1902, Kuliabko, reestablished cardiac contractions that lasted more than one hour in a child 3 months of age, who had been dead for twenty-four hours. This was done by injecting Lock's solution directly into the carotid toward the heart. Crile has restored cardiac pulsation after thirty minutes in a dog by direct blood transfusion through the carotid

toward the heart. Direct injection of fluid into the cavity of the ventricles has been practiced by Duval, Gutig, Hesse and others, but the contraindicatives of this procedure outweigh the questionable advantage of fluid being introduced directly into the cavities over that of simple intravenous injection.

Bandaging of the limbs and compression on the abdomen by forcing the blood through the heart and elevating the pressure in the coronary arteries is of value.

Heydloff reported an instance of cardiac failure in a patient during a cesarean operation. Cardiac massage, artificial respiration and finally the injection of 2 cc. of epinephrine (1:1,000) directly into the heart muscle caused the heart to beat again. From five to six minutes elapsed till the heart again began to beat. The patient remained unconscious for twenty-four hours, but entirely recovered. The state of unconsciousness that immediately results from cessation of the heart beat is apparently due to cerebral anemia, and permanent damage is soon done to the nerve centers if the circulation is not quickly restored. In many of the reported cases the heart has been made to function, but unconsciousness has persisted and the patients have ultimately died from injury to the central nerve. Ten minutes is apparently about the limit that can elapse after arrest of the heart beat without the injury to the central nerve being so great that it cannot recover.

The heart muscle itself is most resistant, and is almost the last tissue to die, but the nervous system is rapidly affected by cessation of the circulation. If the circulation is maintained by intravenous injection of saline, Wrédé has demonstrated that an hour and a half after the heart had ceased to beat, rhythmic contractions were restored and the patient lived three days, but did not recover consciousness. Because of the incidence of cardiac failure during anesthesia, asphyxia and shock from any cause, the value of immediately employing all three or four resuscitory measures, namely, intravenous injection, injection of epinephrine, cardiac massage and bandaging of the limbs, should be borne in mind.

Intensive effort to restore the heart action in sudden failures should be maintained from one-half to one hour at least.

Lenormant has reported twenty-five cases of reanimation via the thoracic route: sixteen times with twelve failures (75 per cent); three times with temporary recovery (18.8 per cent) and once with complete recovery (6.2 per cent), and this via the subdiaphragmatic approach.

Ricketts reports thirty-nine cases. In twelve patients, the heart recovered entirely (34 per cent). In one patient, the heart had ceased to beat for twenty minutes. The ages varied from 22 to 26 years.

In 1914, Tuffier reported fifty-five cases, including those of Mocquot, in which the following methods of approach were used: thoracic route, sixteen with one recovery; transdiaphragmatic, nine with one recovery; subdiaphragmatic, twenty-four patients with thirteen deaths and eleven recoveries—making a total of forty-two deaths and thirteen recoveries.

Mauclaire reported sixty-eight collected cases including those of Cacovic, Mocquot, Juratz, Carleton, Bochus, Aubert, White, Wrédé and Weitz. The sixty-eight cases included twenty-six cases in which the thoracic route was used with death in twenty cases; temporary recovery in four and complete recovery in two; fourteen cases in which the transdiaphragmatic route was used with death in eleven cases and temporary recovery in three; twenty-eight cases in which the subdiaphragmatic route was used with death in six and temporary recovery in nine and complete recovery in thirteen cases.

The failures have occurred in patients in whom the arrest was due to coronary or pulmonary emboli and in patients whose heart showed myocardial changes of long standing at autopsy. In most patients, the failures are due to delay in undertaking massage, and perhaps in addition to not instituting the other measures as pointed out, thereby permitting injury to the central nerve from which the patient does not recover.

Immediately after cessation of the heart beat, at once begin artificial respiration, compress the heart over the precordial area, give the patient sharp slaps with the open hand over the area to excite contraction reflexly if possible, inject 10 minims (1:1,000) of epinephrine directly in the ventricle, elevate and bandage the limbs, and if contractions do not immediately begin, hurriedly open the upper part of the abdomen and begin massage. In the meantime, have an intravenous injection of saline started.

Personal experience has entirely persuaded me that avoidable deaths frequently occur due to lack of appreciation on the part of the medical attendant of the value of cardiac massage associated with all other resuscitory measures. During the Great War when large numbers of patients with grave injuries of all types were constantly under surgical care, sudden arrest of the heart was all too common. If artificial respiration, elevation of the limbs and direct needling of the ventricle or injection of epinephrine into the heart did not cause the heart to function, massage through the subdiaphragmatic route was immediately begun except in cases of pulmonary embolus. One came to realize that massage did not restore life in the latter group of cases. The establishment of cardiac massage in a patient, in whom the heart had ceased to beat for from ten to twelve minutes, again established cardiac function, and the patient recovered. Fortunately, in this

TABLE 2.—*Wounds of the Right Ventricle*

No.	Operator	Year	Location and Character of Wound, Age and Sex	Time Between Wounding and Operation	Position and Size of Heart Wound	Operative Procedure Anesthetic, Sutures	Course and Postoperative Complications	Results
1.	Boussenat.....	1914	Bullet	1 year	.....	Ventricle incised, silk sutures	.....	Recovered
2.	Birkbeck, Lorimer and Gray	1915	Bullet	15 days	Back of heart in either wall or cavity of the right ventricle near apex	Local anesthesia, bullet removed with forceps, wound closed by suture	Four hours after, respiration rose to 48	Died 5 days
3.	Dujarier.....	1916	Fragment shell	.....	.....	Extraction	.....	Recovered
4.	Desplas and Chevalier	1916	Bullet	.....	.....	No operation	....	Death
5.	Debet.....	1917	Man, 31, grenade splinter	70 days	.....	Pericardium incised, no. 2 catgut sutures	.....	Recovered 12 days
6.	H. G. Wood and S. Streight	1918	Male, 19, foreign body; flat piece of shrapnel shell casing	End of 24 hours	Wall of right ventricle	Pericardial sac opened, no foreign body found, closed up, general anesthetic	Entered lower margin of liver, inferior vena cava carried to auricle and into the ventricle, caused terrific cardiac spasm	Death in a few days, autopsy showed piece of shell casing thickness of size tip of little finger
7.	Sir Henry Gray...	1918	Gunshot wound	.....	Right ventricle	Local anesthetic, operation, missile not located	Death 4 to 5 days later, autopsy showed piece of shell casing	Death
8.	Barbier and Gujon	1918	Bullet	23 days	Posterior wall of heart, midway between apex and base in wall of right ventricle	Clamped heart between fingers, 1 suture above and 1 below projectile, incised myocardium and extracted bullet	....	Recovered 30 days
9.	H. Rouvillois.....	1919	Man, 23, Spitz bullet	4½ years	.....	Duval midsternal operation, bullet mobilized with bone scraper, extracted with forceps	....	Recovered
10.	Jenckel.....	1920	Bullet	6 months	.....	First operation, could not bring heart forward; second operation, 6 months later, removed bullet	....	Recovered
11.	Hartman.....	1920	Bullet	2 years, 5 months	.....	Incision and extraction of ball	.....	Recovered
12.	Davenport.....	1924	Stab	About 1 hour	.....	Ligated intraventricular branch of left coronary artery and vein	....	Recovered, out of bed in 13 days
13.	R. L. Rhodes.....	1924	Man, 16, stab	Approximately 1½ hours	Stab passed through pleura, pericardium, right ventricle	Cardiorrhaphy, silk sutures	.....	Died on table, excessive loss of blood
14.	C. Lenormant....	1926	Man, stab wound with awl	1½ hours	.....	Chloroform, resection of ribs, pericardium irrigated and sutured	Pulse fast, temperature elevated for two days	Recovered
15.	P. Fiori.....	1926	Man, 21, needle puncture, left side of chest	About 19 hours	Right ventricle	Exposure of pericardium, needle removed, no record of suture	Effusion into left pleura and pericardium	Recovered

TABLE 3.—*Wounds of the Left Ventricle*

No.	Operator	Year	Location and Character of Wound, Age and Sex	Time Between Wounding and Operation	Position and Site	Operative Procedure Anesthetic, Sutures	Course and Postoperative Complications	Results
1.	H. B. Gessner.....	1912	Man, 24, bullet wound in chest	5 days later	Lower lobe left lung, heart and left ventricle	Introduced fine trocar and cannula, no fluid, ball in cavity of left ventricle	Lobar pneumonia, hemothorax	Death same day
2.	A. L. Lockwood..	1917	Man, 19, missile in right side of chest lodging in left ventricle, hemothorax	14 days	.....	Local anesthesia, missile felt in heart muscle, left in situ	.....	Recovery good, returned to duty
3.	Maj. H. H. Sampson	1919	Bullet wound in left side of chest	.....	Linear tear in pericardium, gutter wound left ventricle	Sutured	Wound healed by first intension	Recovery
4.	Sir Berkeley Moynihan	1920	Shrapnel ball wound in left midaxillary line	12½ months	Between auricle and ventricle, embedded in wall of left ventricle	Pericardium opened, ball removed with scoop, two sutures	Speedy recovery but unable to do usual work owing to lung injury	Recovery
5.	J. F. X. Jones....	1922	Man, 33, stabbed with long, slender knife	1 hour 10 minutes	Left pleura punctured near apex extending into left ventricle	Wound closed with two catgut sutures	.....	Died on table
6.	J. H. Long.....	1925	Man, 45, stab wound entered pericardium and pleural cavity	.....	Left ventricle	Ether anesthetic, wound in muscle of left ventricle, sutured with one chromic gut	Uneventful	Recovery
7.	I. M. Bufalini.....	1926	Four stabs involving heart	.....	Left ventricle	.....	.....	Recovery
8.	I. M. Bufalini.....	1926	Two stabs	.....	Wound in anterior face of left ventricle, another in lobe of liver	.....	.....	Death
9.	J. L. Fisher.....	1926	Man, 24, stabbed with pocket knife	45 minutes	Laceration of left ventricle	Ether, four silk sutures	Friction rub, lung involvement	Recovery
10.	W. H. Cole.....	1927	Man, 30, stabbed in anterior part of thorax, broken blade left in wound	.....	Wound in left ventricle	Local anesthetic and ether, suture	Pneumonia for 10 days; abscess right clavicle	Recovery in 6 weeks

patient saline was being administered intravenously to offset the effect of hemorrhage when the heart ceased to beat. Artificial respiration, hypodermics and dilatation of the rectum did not excite the heart to contract, and all effort to resuscitate the patient was about to be abandoned. Within two minutes, after hurriedly opening the abdomen and starting direct cardiac massage, the heart began to contract, and after twenty minutes the patient regained consciousness and recovered. With this recovery always in mind, I have never hesitated over from three to four minutes to begin direct cardiac massage through the subdiaphragmatic route. Recently, the heart suddenly stopped beating in a young woman eight hours after a simple appendectomy. Approximately from eight to ten minutes after the heart had ceased to beat, massage was begun. The patient had passed from the blue stage of cyanosis to an ashen gray pallor. For at least one minute after massage was begun the heart refused to contract; then faint contractions developed; within from three to four minutes the heart was contracting irregularly about 120 times to the minute. Color returned to the face and mucous membranes, but the pupils remained dilated and respirations did not recur. The heart continued to contract for one hour with periodic massage and then ceased in spite of the use of a pulmotor, intravenous injection of saline and direct injection of epinephrine into the heart muscle. Undoubtedly, had some one been at hand to have started massage earlier before too extensive injury to the central nerve had occurred, this patient might have recovered. Unfortunately, autopsy was not permitted, but an experienced nurse and intern were firmly of the opinion that death was not due to pulmonary embolus.

It is most essential to commence massage without delay. Do not let the danger of peritonitis delay opening the abdomen till it is too late to profit by the massage.

Paracentesis of the Right Auricle and Ventricle.—I merely mention this procedure to condemn it. The needle is passed through the third right intercostal space close to the sternum to enter the right auricle, and then the fourth right intercostal space 1 inch from the sternum to puncture the right ventricle. Suction is necessary to withdraw blood from the auricle because of the low pressure within it. Venous section of the external jugular vein will accomplish the same result as paracentesis.

Operation for Pulmonary Embolism.—Trendelenburg's operation for removal of blood clot from the pulmonary artery is an heroic measure, yet I am sure that most surgeons who perform a large number of operations have this procedure frequently in mind. A "T" incision is made from well above the second cartilage to the fourth and extending laterally below the second rib for from 10 to 12 cm. The

TABLE 4.—*Wounds of the Right Auricle*

No.	Operator	Year	Location and Character of Wound, Age and Sex	Time Between Wounding and Operation	Position and Site	Operative Procedure Anesthetic, Sutures	Course and Postoperative Complications	Results
1.	Capt. John Fraser	1917	Wound in chest	.....	Valvular perforation upward and inward ⅛ inch in anterior wall of right auricle	Pericardium incised, heart exposed, traction sutures, two mattress sutures	Progress uneventful	Recovery
2.	R. L. Rhodes.....	1924	Man, 23, stabbed in left side of chest with scissors	1 hour	Wound in right auricle	Ether, pericardium opened, blood evacuated, heart wound sutured	Temperature normal on fifteenth day when patient developed tonsillitis	Recovery
3.	D. McGuire.....	1925	Man, 18, stab wound in chest	1½ hours	Wound in right auricle	Ether, incision over sternum, opening in pericardium enlarged, sutured chromic gut no. 1	Transfusion of blood, stormy convalescence	Recovery

TABLE 5.—*Wounds of the Left Auricle*

No.	Operator	Year	Location and Character of Wound, Age and Sex	Time Between Wounding and Operation	Position and Site	Operative Procedure Anesthetic, Sutures	Course and Postoperative Complications	Results
1.	Hamilton Drummond	1925	Man, 25, stabbed with penknife	Several hours	Hole at base of pericardium, tip of left auricle wounded	Ether, left pleura opened, wound of auricle closed with catgut suture	Pleural effusion, blood aspirated 14 days after operation, empyema, left side of chest drained	Recovery

second left cartilage and a portion of the rib are excised. The third cartilage is severed close to the sternum. The internal mammary artery is tied, the pleura displaced laterally and the pericardium opened. By some selected and previously well perfected plan, the circulation through the aorta and pulmonary artery must be arrested for not more than one and a half minutes. I believe it is best done with two rubber covered clamps shaped like the curved flat intestinal clamps, but smaller. If preferred, a small Carrel tube, as suggested by Trendelenburg may be carried behind the aorta and the pulmonary artery, to control hemorrhage. When the pulmonary artery is incised and the clots removed, one mattress suture of fine petrolatum silk on a no. 15 curved Irwin needle is hurriedly passed and tied, closing the incision. Then by a select type of forcep, the pulmonary artery is grasped so as to have the incision in the forcep. The clamps are released and the suture of the incision in the vessel is completed. The procedure of incising the artery, removing the clot, inserting one suture and placing the forcep on the pulmonary artery requires from thirty-five to forty-five seconds. I have carried out the procedure repeatedly at autopsy. It is not at all a difficult feat. Although I saw many deaths from pulmonary embolus during the Great War, and unfortunately a number since, and although since the fall of 1916 I have planned to undertake this procedure when the opportunity occurred, I have yet to see a patient early enough to hope for any benefit from operation. In two cases with recurring emboli, in which everything was ready for operation should an apparently fatal embolus occur, the patients recovered. Undoubtedly, theoretical surgical intervention is rational. Unfortunately, however, the surgeon is rarely at hand early enough to hope for any benefit. I have always felt that Trendelenburg was fortunate (paradoxically speaking) in having seen twelve patients dying from pulmonary embolism sufficiently early to permit of operation. None of these patients survived. Professor Kirschner (1924, Konigsberg), following Trendelenburg's technic, was the first to succeed in saving a life by this means. Schumacher had one patient who survived for five days, and finally died of pneumonia. Kruger had a similar case. Four successful cases demonstrate that the operation is practicable. Great care and experience are necessary to determine the moment to undertake what may of itself be a fatal procedure in a patient who might have recovered spontaneously. Every surgeon should be equipped and prepared to undertake such an operation should the occasion arise.

The extrapericardial route as carried out by Neuhof in a woman, aged 40 (moribund), deserves consideration. It is a question if through a modified Kochre incision as suggested by Lilienthal such an operation could not be more successfully carried out.

Paracentesis and Pericardiotomy.—In 1649, Riolanus first advocated paracentesis pericardii. In 1818, Skielderup trephined the sternum for paracentesis, and in 1871, Malle performed the same operation. Dieulafoy advocated aspiration rather than the radical method of Riolanus. Trousseau (France) practiced open incision of the pericardium. In 1866, Wheelhouse did a paracentesis on a patient of Clifford Albutts. The patient was in extremis, but improved immediately and recovered. Shortly after, Bowditch, in the United States, Roberts of Manchester, England, and several others reported cases. In 1876, John B. Roberts reported forty-one collected cases.

While some successful cases have been reported in doing the paracentesis, wounds of the heart, the coronary arteries and the internal mammary have occurred. In addition, one of the main objections is the danger of carrying the infection into the lung or pleural cavity, thereby setting up another serious condition. Ballance expressed the opinion that the operation of paracentesis pericardii should be banished from surgical practice. He advised removing the sternal end of the left sixth and seventh cartilages. The internal mammary artery must be drawn aside and tied and the triangularis sterni divided.

Concensus of opinion is strongly in favor of pericardiotomy in preference to paracentesis.

In 1922, Hedbloom published a complete resumé of this subject to that date.

The first successful pericardiotomy was done by Romero of Barcelona, in 1819. He operated on three patients with two recoveries. He incised the chest in the fourth and fifth interspaces near the left border of the sternum. In 1844, Hilsman reported a successful operation. In 1881, Rosenstein did the first rib resection for drainage. In 1897, Voinitch published a comprehensive article on the relations of the pleura to pericardium and advised section of the sixth and seventh costal cartilages. In 1897, Porter of Boston collected twenty-four cases and added a successful case of his own. Death occurred in sixteen of the cases, recovery in eight and the results in one are not known.

In the same year, Roberts reported thirty-five cases with recovery in fifteen and death in twenty. Ljunggren (1899) reported forty-one cases with recovery in sixteen and death in twenty-five. In 1900, Porter reported fifty-one cases with recovery in twenty and death in thirty-one. Eliot (1909) added twenty-two cases. In 1915, Rhodes reported thirteen additional cases, and in 1921, Poole added thirteen collected cases. In 1927, Williamson added eighteen cases, making a total of 117, including those reported by Porter, Eliot and Rhodes. Of the 117 cases, recovery occurred in sixty-three and death in fifty-four, or 54 per cent cures. Klose and Strauss reported ninety-three collected

TABLE 6.—*Miscellaneous Cases*

No. Operator	Year	Location and Character of Wound, Age and Sex	Time Between Wounding and Operation	Position and Site of Heart Wound	Operative Procedure, Anesthetic, Sutures	Drainage	Course and Postoperative Complications	Results
1. W. E. Lower.....	1916	Rifle ball	3½ months	Point of bullet had passed through pericardium	Nitrous oxide anesthesia, bullet removed, mattress sutures of chromic gut	Small cigaret drain in pericardium	Developed pneumonia followed by effusion	Complete recovery
2. A. L. Lockwood..	1916	Man, 26, shrapnel left of sternum in fifth interspace	.....	Laceration of pericardium with missile projecting into pericardial sac	Wound excised, missile removed, ecchymosis over right ventricle but no wound suture of pericardium	.....	Healed by first intention, uninterrupted convalescence	Recovery
3. Maj. Littler Jones	1917	High explosive shell	8th day	Deep groove in upper border and anterior surface of the vent	Resection, adhesions separated	.....	Wound healed in 7 weeks	Recovery
4. Maj. Scott-Skirving	1917	Fragment of shell in posterior axillary line, age 22	About 5 months	.....	Pericardial cavity incised, foreign body removed	.....	Uninterrupted recovery	Recovery
5. Capt. W. Briggs..	1917	Bullet entered right posterior axillary line	9th day	One inch above cardiac apex	Pericardium incised, bullet removed from wall of heart	.....	Severe pericarditis	Died 8 hours later
6. Escande and Brocq	1917	Bullet inside cardiac shadow	.....	Embedded in myocardium	Patient tolerated bullet, no operation	Pericardium animated by regular oscillations synchronous with cardiac contraction	.....	Recovery
7. Idem.....	1917	Splinter of shell	.....	In apex of heart	Well tolerated	No operation on pericardium	.....	Recovery
8. A. L. Lockwood..	1917	Man, 37, shrapnel wound left chest, laceration lower lobe of lung, missile embedded in mediastinum-pneumothorax	8 hours	Pericardium torn, no injury to heart	Chest opened, missile removed, pericardium entered	.....	.....	Good recovery
9. Duval and Barasty	1918	French soldier, German Spitz bullet in left side of thorax	.....	Bullet in inferior vena cava	Midsternal operation	Foreign body extracted with forceps from pericardium	Pericardial friction, small pulmonary infarct	Recovery
10. G. Gillen..... (2 cases)	1918	Gunshot wound left chest laceration pericardium	.....	.....	Pericardium entered in the course of a thoracotomy	.....	.....	One died, one recovered
11. Sir Berkeley Moynihan	1920	Fragment of shell	.....	.....	Third left rib raised, foreign body removed	.....	.....	Well in 2 years
12. Idem.....	1920	Shrapnel ball	2½ years		Pleura opened, adherent lung peeled from pericardium and ball removed	.....	.....	Fit and well in 1 year and 8 months

13. Idem.....	1920	Bullet left side of chest	2 months	Bullet imbedded in fatty fibrous tissue adherent to surface of heart	Pleura opened, adherent lung peeled from surface of pericardium	.....	Bullet sterile on incubation	Recovery
14. Idem.....	1920	Shrapnel ball behind sternum	.....	.....	Pericardium penetrated, ball lifted from fibrous sac	.....	Uneventful	Recovery
15. W. H. Luckett....	1920	Woman, 19, bullet wound	8 days	Right side heart near apex	Nitrous-oxide and ether anesthetic, incision through right ventricle, missile extracted, catgut suture	Drainage of pericardium	Stormy convalescence	Recovery
16. C. Goodman.....	1920	Man, 46, stab wound	1 hour	.....	Exposed heart, entered pericardium through pleura	Drainage of pleura	Sepsis incident to suppurating pleuritis	Death 1 week later, sepsis attributed to drainage of pleural cavity
17. E. M. Frese.....	1921	Stab wound	1 hour	Both ventricles	Simple suture	.....	.....	Recovery
18. Claude S. Beck and R. L. Moore	1923	Boy, 13, stab wound in 4th interspace	Immediately	.....	Ether, pericardium opened, blood evacuated, no wound found in heart, silk sutures, (wound may have been sealed with clot)	Rubber drain in pericardium	Profuse bleeding from stab wound, opened and explored, a few sutures removed from pericardium	Recovery
19. H. Klose.....	1926	Man, 23, bullet wound in posterior wall of thorax	32 hours	Wound in heart 2 inches long	Left index finger inserted until papillary muscles of valve felt, closed with five sutures	.....	Extra systoles first five days	Recovery good
20. G. P. La Roque...	1926	Man, 17, bullet wound left side of back	12 days later clotted blood removed, two mos. later operation for relief from blockage of femoral	Fragment of bullet in left side of chest, remainder 2 inches below Poupart's	No wound of artery, permanent silk ligature of vein and bullet excised	.....	No pulse below lesion either before or after operation	Recovery
21. J. C. O'Day.....	1927	Fragment of emery wheel in left side of chest puncture wound	2 hours	.....	Suture with iodized silk	.....	Uneventful	Recovery
22. Idem.....	1927	Stab wound, upward thrust	6 hours	Pericardium pierced	Pericardial sac slit and blood turned out, cut shallow, no suturing	.....	.....	Recovery

cases with the following results: of twenty-nine patients treated by puncture only, four recovered; of twenty-seven treated by intercostal incision and drainage, ten recovered; of thirty-seven treated by wide exposure, twenty-one recovered. Whitmore (Boston) reported a successful recovery after incision and drainage by the closed suction method.

Larrey published a case of paracentesis of the pericardium and later of pericardiotomy. He planned the operation taken up by Allingham ninety years later.

Larrey operated on a man 30 years of age, who had stabbed himself in the heart forty-five days before. The incision was made in the fifth interspace below the nipple. He incised the tense pericardium with a bistoury and inserted his finger and palpated the apex of the heart. Almost a liter of blood-stained fluid and blood clot escaped with immediate relief. The patient improved and in ten days the

TABLE 7.—*Pericardotomy*

No.	Operator	Year	No. of Cases	Site of Puncture	Death		Recovery	
					Number	Per Cent	Number	Per Cent
1	C. B. Porter.....	1900	51	.....	31	60.9	20	39.1
2	Salomoni.....	1901	36	.....	13	36.1	23	63.9
3	Reichard.....	1901	33	.....	19	57.1	14	42.9
4	E. Eliot, Jr.	1909	22 (since Porter's report)	.....	7	31.8	15	68.2
5	Jacob and Roehard	1913	2	.....	..	....	2	100
6	J. C. O'Day.....	1927	1	Liberal slit in pericardium with free drainage	..	....	1	100

wound healed. The symptoms of distress recurred. The wound was opened with a probe and 6 ounces of pus escaped. Considerable improvement resulted. Death from sepsis ensued twenty-three days after operation and sixty-eight days after injury. Autopsy revealed a suppurative mediastinopericarditis. Larrey carried out various operations on cadavers. He was prompted to carry out this procedure when treating a man who had stabbed himself under the seventh rib. There was a hemopericardium with considerable blood clot. Larrey evacuated the contents of the pericardium and inserted a catheter. The patient had a stormy convalescence, but recovered.

Allingham's so-called "epigastric operation" for drainage of the pericardium is similar to that suggested by Larrey. He developed it on twelve cadavers, but never applied it to the living. It establishes free access to the sac and dependent drainage from below through the diaphragm.

An incision 2 inches long extends from the costoxiphoid angle along the lower edge of the seventh costal cartilage. The abdominal muscles are reflected from the edge of the rib and the cartilage retracted

up and out, exposing the diaphragm with the cellular area between its attachments to the cartilage and to the xiphoid appendix. This cellular space is exposed by splitting the diaphragm as far as necessary. A pad of fat presents itself with the diaphragm below, the sternum in front and the pericardium above and behind. By retracting the diaphragm and fat downward the pericardium is exposed, palpated and opened dependently. While the peritoneum is exposed as it is reflected downward from the under surface of the diaphragm, there is no occasion to injure it. It is merely packed off. The pleura cannot be opened. A free exposure of the pericardium is ensured except in obese patients, and dependent drainage is established. If the patient's condition will permit it, I believe this is the operation of choice for drainage of a purulent pericarditis. Many of these patients are gravely ill, and the procedure of dividing the fifth and sixth costal cartilages close to the sternum and thus exposing the pericardium is quicker and more easily borne, but dependent drainage is not so completely secured. Matas has pointed out that the lower the site of drainage of the pericardium the better, and the less the risk of injuring the pleura or the heart.

Trousseau, in urging pericardiotomy in preference to paracentesis, relates the interesting case of Roux, in which Roux, after exposing the pericardium, palpated it with the finger and found the heart to be in contact with the pericardium. He could not find an area of fluctuation and closed the incision without incision of the pericardium. Death ensued and autopsy revealed a "dilatation phénomenale" with attenuation of the wall of the heart. There were no valvular lesions and no serosity in the pericardium.

Delorme and Mignon advised a vertical incision from the lower border of the seventh costal cartilage to the upper border of the fourth cartilage, 1 cm. external to the left border of the sternum.

Porter advised excision of the fifth costal cartilage. This is probably the operation of choice, if the patient is seriously ill. It can be carried out with the least disturbance to the patient. I have successfully drained the pericardium in one case through this route, and in one patient after resection of the sixth and seventh costal cartilages.

Major Littler Jones, in 1916-1917, published a most interesting case of the soldier hit by shrapnel in the precordial area. Both wounds were infected, and the patient had a stormy eight days, the condition presumably being due to double hemothorax. On the eighth day, the pulse was 70, the temperature was normal and the wounds were clean. The face was dusky and the patient had a constant stretching pain through the left side of the chest posteriorly. Fluoroscopic examination of the chest revealed a greatly distended pericardium and little fluid in the pleural cavities. Half the width of the sternum and the

fifth, sixth and seventh costal cartilages were resected. The pericardium bulged into the field and on opening it 22 ounces of foul-smelling fluid escaped. There were some adhesions between the parietal and visceral layers of the pericardium and a deep gutter wound in the upper and anterior surface of the right ventricle. The heart beat immediately improved on opening the pericardium. The pericardium was drained and irrigated with hypertonic saline, eusol and iodine water. The patient recovered. Thirteen weeks later, he had regained his previous good health. *Bacillus perfringens* and enterococci were cultivated from the fluid removed.

The insidious onset of anaërobic infection without marked febrile disturbance up to a point is important. The relief afforded and the

TABLE 8.—*Cardiolysis for Adherent Pericarditis*

No.	Operator	Year	Reporter	No. of Own Cases	No. of Collected Cases	Death	Recovery
1	Peterson and Simon	1902	Ballance	3	3	1	2
2	Lacheme.....	1909	Babcock	...	20	...	20 relieved
3	Thorburn.....	1910	Thorburn	1	18	...	18 (12 encouraging, 6 relieved)
4	Delagenière.....	1913	Delagenière	...	38	1	37 (failures; 31 relieved)
5	Couteau and Bellot	1916	Official History of the War	1	1	1	
6	Lockwood.....	1919	.....	1	..	...	1 relieved
7	G. Bourne.....	1924	Bourne	2	25	...	
8	Marvin and Harvey	1924	Marvin and Harvey	1	11 (not included in Bourne list)	...	
9	Loe.....	1925	Loe	1	1	...	
10	J. A. Mattison.....	1927	J. A. Mattison	1	1	...	1

return to health following operative intervention in such a type of infection is most interesting.

In 1919, Sir George Makins reported two cases in which he drained the pericardium for purulent pericarditis following gunshot wounds. He resected the sixth and seventh costal cartilages.

Adequate drainage of the posterior compartment of the heart and particularly the pouch along the transverse sinus is most difficult. Balance has stressed the necessity of draining this area. In order to gain access to this area, it is necessary to open the pericardium widely and to clear out the pus thoroughly, by either suction or careful sponging. Autopsy examination reveals the fact that pus is always retained posteriorly if drainage has not been adequate (fig. 1). It is questionable if the heart itself will as a rule tolerate a tube carried behind it into the pouch of the transverse sinus. The heart in Whittemore's patient was apparently not disturbed by the tube, but generally speaking, I believe that if the posterior wall of the heart came in contact

with the tube with each beat of it, it would act exactly as a foreign body, and excite such a tumultuous beat that it would do more harm than good. Experimentally, it is difficult to drain this area on cadavers. For that reason I believe dependent drainage through the lowest accessible portion of the pericardium should be secured, the pericardium should be carried out and sutured to the skin and careful irrigation through a two way rubber catheter should be employed, if necessary, being guided by the febrile state of the patient.

Unquestionably, in purulent pericarditis or even serous pericarditis with marked distress and a febrile reaction, dependent pericardiotomy

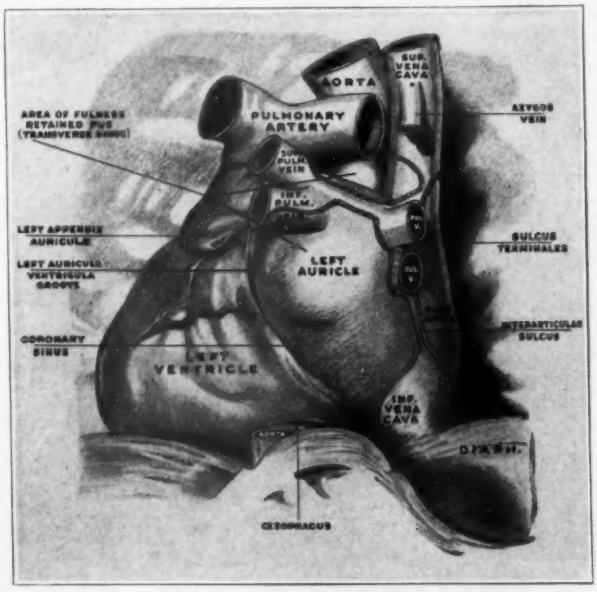

Fig. 1.—Base of the heart in place. Note the fulness above the transverse sinus and behind the left auricle, due to retained pus.

should be undertaken without delay. With local anesthesia and a careful approach, the risk of pericardiotomy will be less than that of paracentesis, and I believe with approximately a 60 per cent chance of cure. Autopsy observations reveal that this grave condition is all too frequently overlooked and that even when diagnosed the patient has not been given the chance of obtaining a surgical cure.

Cardiolysis.—This operation is planned to remove the bony wall of the chest overlying the heart and release pericardial adhesions, should they be sufficiently binding to prevent free movement of the heart after removal of the compressing wall of the chest. It is a wonderful operation in selected cases. Delorme (1898) and Weil of Lyons (1895),

advised that all the adhesions anteriorly between the parietal and visceral layers of the pericardium should be free surgically. This is not a simple procedure, and should not be undertaken unless it is evident that the heart is so bound by pericardial adhesions that removal of the wall of the chest is not going to benefit the patient. In 1909, Leriche approved of the Delorme procedure, and suggested that in addition, excision of the pericardium should be done. Personally, I cannot easily approve of this procedure. Ordinarily, removal of from three to four cartilages and a portion of the sternum, if necessary, is sufficient. Peterson and Simon, at Brauer's suggestion, first performed this operation in 1902. In 1910, Thorburn of Manchester reported a case of his own and two by Stabb. He collected fifteen cases reported in the literature. Alexander Morrison reported one case in which operation was performed to allow more room for an enlarging heart in a case of aortic stenosis. In Thorburn's eighteen collected cases, six patients were greatly improved, and in others the results were encouraging, but details were not recorded.

In 1924, Bourne collected twenty-three recorded cases and added two of his own in which he did a cardiolysis. Marvin and Harvey reported a case the same year, making only six reported in the United States. They collected ten cases reported by Treupel, Soyesima, von Jagic, Thorburn, Dunant and Turettini, Ensgraber and Schwarzenauer and three reported by von Beck, which were not included in Bourne's series.

Summers, Dunn and Summers, Hirschfelder, Smith, Marvin and Harvey, Loe, and Mattison each reported a case, making seven from the United States.

I have employed this operation on only one patient. This patient had an adherent pericarditis and a bad bony deformity involving the fourth, fifth and sixth cartilages on the left, following a gunshot wound received four years before. The third, fourth, fifth and sixth cartilages were excised, and about 1 inch of the sternum was removed with bone nibblers. The pericardium was firmly adherent to the heart, but immediately the whole heart seemed to move forward. The operation was done entirely under local anesthesia. The patient stated that he could breathe more freely before he left the operating room. The operation was performed in 1919. The patient was alive and well, and much better when last heard from in 1922.

I am fully convinced that this operation should be more frequently employed, not only on patients with adhesive pericarditis, but also on patients who have marked enlargement of the heart in a fixed type of chest with edema of the limbs and other evidence of cardiac decompensation, not directly due to aortic regurgitation.

Cardiorraphy.—The selection of the type of operation depends on whether the operation is urgently required and must be hastily performed or may be delayed and carried out deliberately; whether a wound of the chest is present that must be excised and dealt with; whether the pleura and lung are involved, the position of the missile and the general condition of the patient. I agree with Tuffier's advice that all wounds of the heart that are bleeding should be operated on and at least explored. The necessity of late operations for removal of

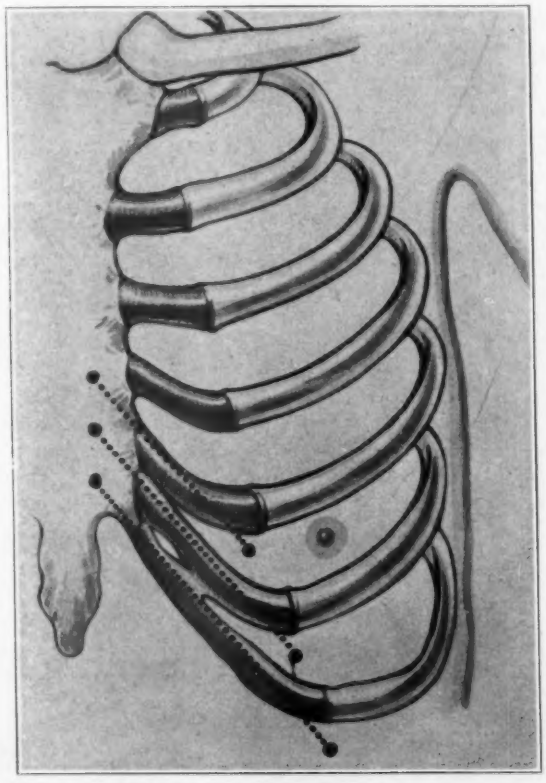

Fig. 2.—Lines of incision for exploratory pericardiotomy which radiate from the left sternochondral angle, including the fifth, sixth and seventh costal cartilages. 1 indicates the incison for resection of the fifth cartilage and edge of the sternum, proposed by Ollier (1891), C. B. Porter and Durand (1896), etc.; 2, the resection of the sixth cartilage and adjoining sternum, proposed by Kocher (1903), Voinitch, etc.; 3, the resection of seventh costal cartilage, proposed by Mintz (1904), Rehm (1907) etc.

retained missiles in the heart or pericardium depends on the severity of the symptoms. The important symptoms are, rapidity and irregularity of the heart's action on exertion or excitement or stress of any kind with dyspnea, precordial pain and postural distress.

Occasional spells of chills with general malaise, tachycardia and a variable febrile reaction occur, exactly as with retained missiles in lung tissue and warrant removal (figs. 2, 3, 4, 5 and 6).

Delorme-Mignon Operation.—Kocher Modification: If the entrance wound need not be followed, the Delorme-Mignon operation, the Kocher procedure or some such modification should usually be chosen. If rapid exposure is necessary, a square flap of the soft parts is reflected outward, the fourth, fifth and sixth cartilages are

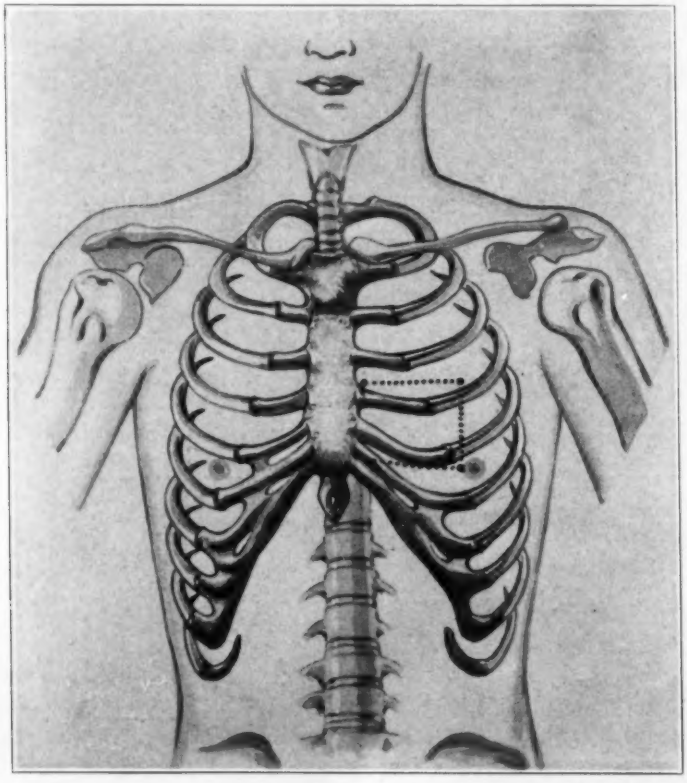

Fig. 3.—Flap of sternum and cartilages of the fourth and fifth ribs; hinge on the right (H. Lorenz and others).

excised, the internal mammary artery ligated or retracted, the triangularis sterni freed and the edge of the pleura and lung retracted and the pericardium then incised freely. Kocher advocated a chondroplastic flap with the hinge outward, which I prefer (figs. 7, 8, 9 and 10).

The Spangaro Operation: Spangaro makes an incision in the fourth or fifth interspace and severs or disarticulates the cartilages of the fourth and fifth ribs close to the sternum. If more exposure is

necessary, the third and sixth cartilages may be cut. In a high percentage of wounds of the heart and pericardium, the pleura and lung are involved. In such instances, this incision is the one of choice. A powerful ribspreading retractor such as Myers', Lilienthal's, Tuffier's, or the author's modification of it, is necessary. Exposure of the left wall and posterior aspect of the pericardium is best secured by this operation. Only in this way can the sac above the transverse sinus, the culdesac of Haller, be readily approached and drained. Lacerations of the lung

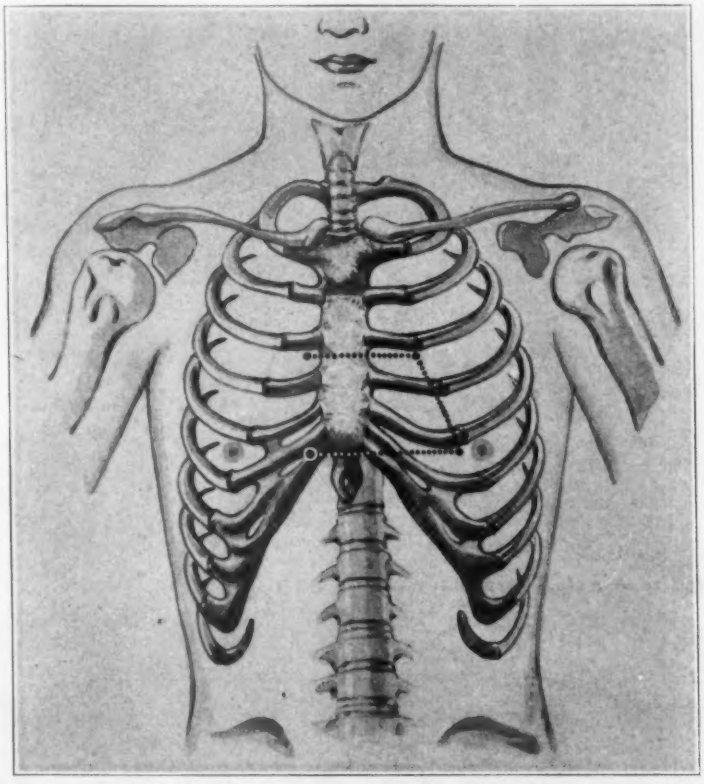

Fig. 4.—Flap of the fourth and fifth ribs and their cartilages; hinge internal.

can be dealt with and the hemothorax and retained blood clot may be manually cleared out of the pleural cavity.

I have employed this incision many times for thoracotomy. It gives a wonderful exposure, and practically any type of intrapleural operation may be readily carried out through it. Care must be taken, however, to approximate carefully the adjacent ribs after removing the retractor. It is not always easy to restore the proper alignment of the ribs, if they have been spread for some time. I have had to bind them together with no. 2 chromic catgut on occasions and have had to

go further and excise one rib at the end of the intrapleural procedure, in order to get an easy approximation of the pleura and overlying muscles which is necessary for the "hermetical sealing" of such an incision in the pleura. This is one objection I have encountered to this incision. In addition, the right auricle and the venae cavae are not exposed and the left auricle is poorly exposed (figs. 11, 12 and 13).

The Duval and Barnsby Operation: This type of operation, independently planned and practiced by Duval and Barnsby, is applicable

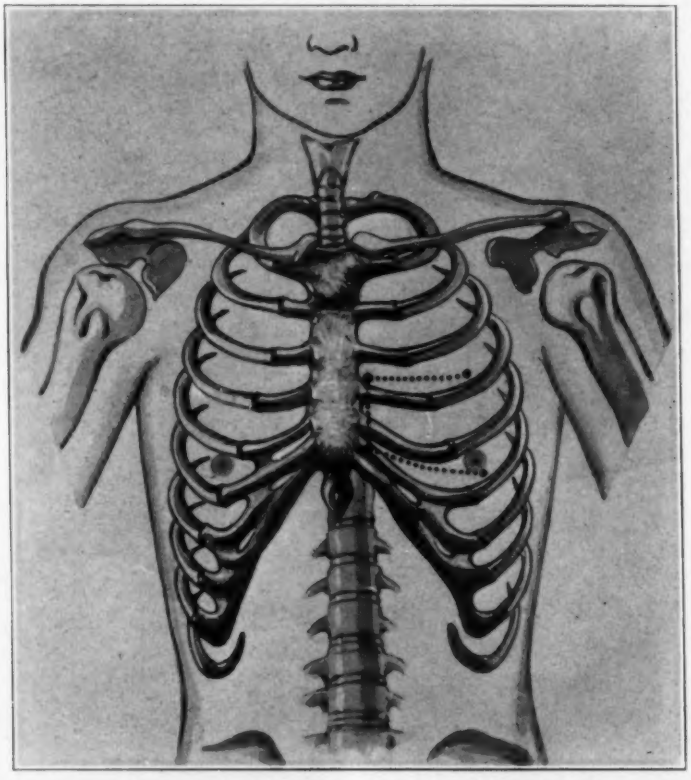

Fig. 5.—Flap of the fourth and fifth ribs and their cartilages; hinge external.

only to conditions in which rapid exposure of the heart is not of paramount importance. It gives an excellent exposure of the pericardium and heart. Duval, in association with Henry and Barasty, described the procedure in detail, classing it as a "median thoraco-abdominal pericardiotomy."

Median Thoraco-Abdominal Pericardiotomy.—A median incision, extending from the level of the third costal cartilage to a point in the linea alba, midway between the tip of the xyphoid cartilage and the umbilicus. After cutting through the skin to the sternum, the incision is carried through the abdominal aponeurosis and between the recti down to, but *not through,* the peritoneum.

The upper insertions of both recti are detached from the edge of the ensiform cartilage, exactly through the median line and the posterior surface of the ensiform, the attachments of the diaphragm also being cut away from the cartilage. Two fingers of the left hand are inserted and pushed from below upward and behind the xyphoid, and then behind the sternum up to the level of the third costal cartilage, the fingers always closely following the posterior surface of the bone.

In this way the pericardium is detached from the retrosternal connective tissue. By separating the fingers slightly, they push and crowd the two pleural cul-de-sacs away from the mid-line and laterally to the under surface of the costal cartilages. This manoeuver is simple and easy.

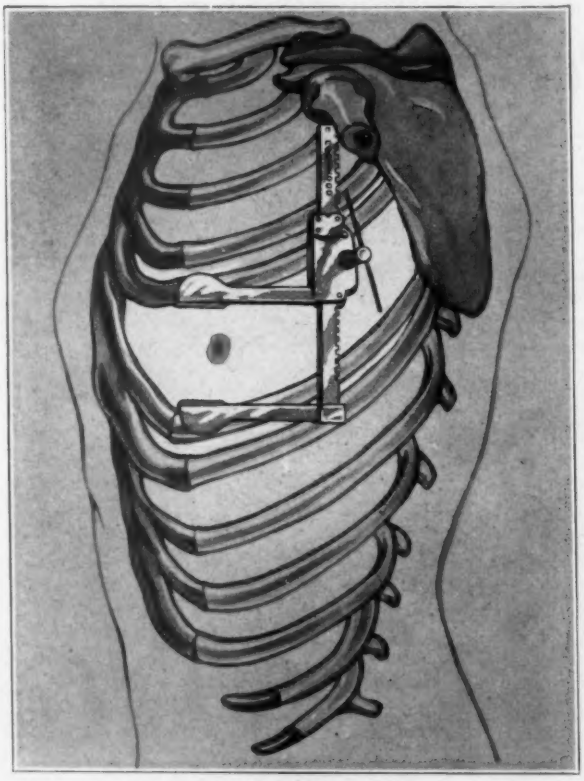

Fig. 6.—Intercostal Thoracotomy.

Longitudinal Sternal Section.—The sternum is then split in the mid-line with a large chisel (Hudson's large sternotome or costotome does better and quicker work), beginning from the tip of the xyphoid and ending on a level with the third costal cartilage. At this level a transverse section of the sternum is made, leaving the manubrium attached to the clavicle and to the first and second cartilages. The two halves of the sternum are now spread open and lifted out, and bent over with retractors or strong hooks, care being taken not to fracture the cartilages. The mere lifting up and spreading of the two halves of the sternum with an outward bending of the yielding costal cartilages will suffice to give a good working space in the mediastinum. Through this large vertical space the pleural cul-de-sacs

and pericardium are recognized, displaced, and detached from the thoracic wall with gentle gauze sponge pressure to avoid any tearing of the pleurae.

Laparotomy and Pericardiotomy.—The peritoneum is now opened in the mid-line. By inclining the incision a little toward the left and immediately along the insertion of the diaphragm to the chest wall the pleurae will be seen to diverge, leaving a very considerable and safe interspace between them. The pericardium is now held up with two forceps and the fold thus lifted up is divided in the mid-line. Each edge of the pericardial incision is now held in a forceps as the heart is exposed.

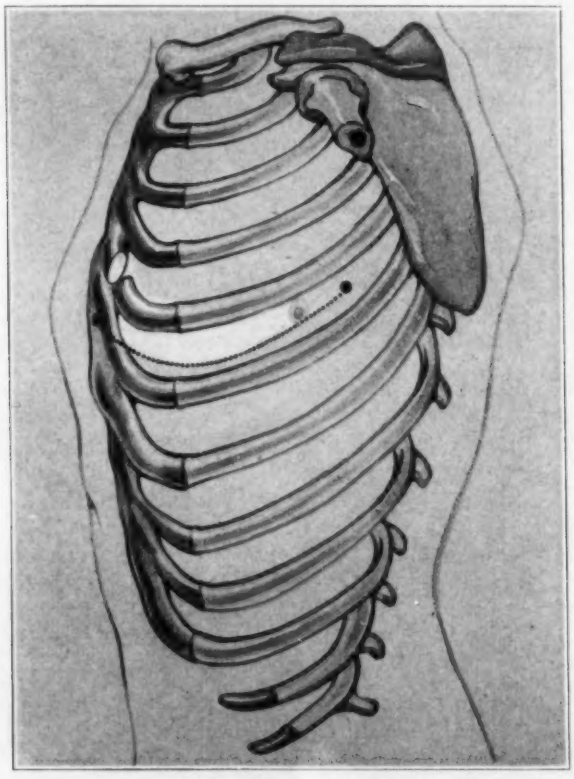

Fig. 7.—Spangaro incision

With sharp, straight, but blunt-pointed scissors the pericardium is now split its full length to the root of the great vessels. The pleural cul-de-sacs are now safe and need not be considered.

Section of the Diaphragm.—Between the pericardial and peritoneal sacs the diaphragm still remains as a horizontal partition holding the two halves of the divided xyphoid in place. The scissors are again introduced and the diaphragm is divided up to the level of the suspensory ligament. While this manoeuver is being executed the heart is gently protected by the hand of an assistant. The hooks or retractors then pull the sternal halves apart, and by lifting them up and bending them outward upon their cartilaginous attachments they widely open the retro-sternal space. It is like opening a book, exhibiting the heart with the

great vessels at its base in full view before the operator. The ventricles, auricles, aorta, and venae cavae, anterior and posterior surfaces of the heart are thus made more easily accessible to any manipulation without requiring exteriorization, traction, or torsion of the organ. The two hands of the operator may execute any manoeuvers or procedures that may be required, with the heart in place.

After the suture of a wound or extraction of a projectile the operation is concluded by the suture of the diaphragm, then of the abdominal incision and of the posterior wall of the pericardium. The sternal halves are replaced and held in contact by deep aponeurotic sutures without bone suture support. Finally a continuous or interrupted suture closes the skin.

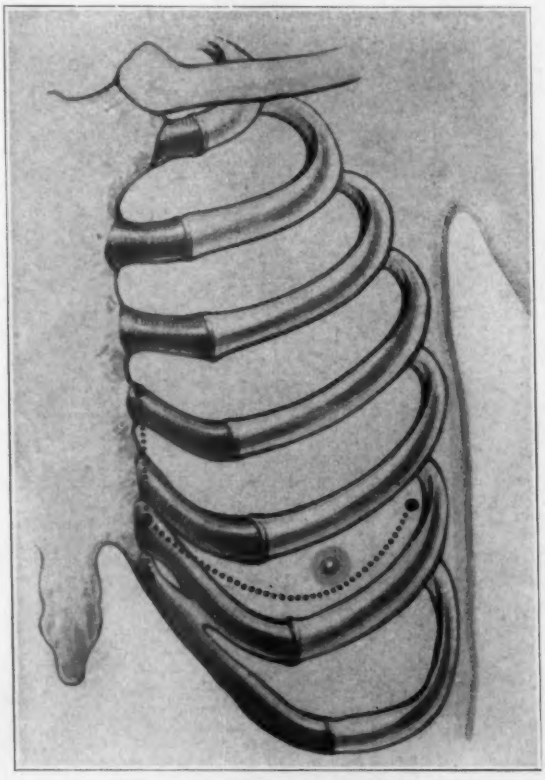

Fig. 8.—Modified Spangaro incision

While this operation at first seems radical and of great magnitude, in reality it is not difficult, dangerous or long drawn. As the incision is confined to the midline, it is almost a bloodless operation. I have employed it in two cases of extensive mediastinal sarcoma with relief from dyspnea and pressure. The procedure of freeing the posterior surface of the sternum from its attachments does not require more than a minute or two. Two fingers are passed up and pressed firmly to the bone exactly as in removing the sternum at autopsy. An electric saw is a

great convenience in splitting the sternum. I have repeatedly carried out this procedure on cadavers, and I am thoroughly satisfied that it is the operation of choice for the following conditions:

(1) Late operations for removal of missiles retained in the heart or pericardium; (2) possibly for a certain percentage of patients with marked distress from mediastinopericarditis; (3) certain types of malformations of the chest with cardiac and respiratory distress and (4) for mediastinal decompression (of course, avoiding in such cases, opening the pericardium).

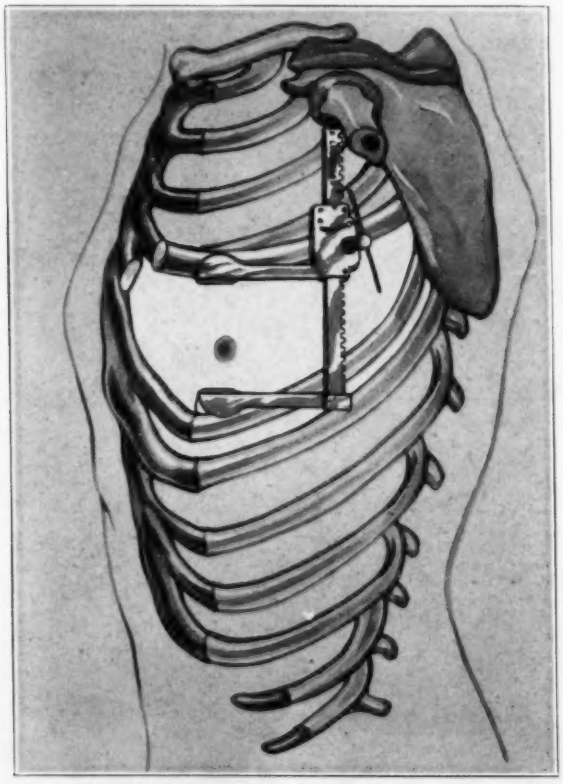

Fig. 9.—Spangaro incision.

As early as 1918 and 1919, Duval and Barnsby, Barbier and Gujon and Rouvillois reported a successful case in each instance following such an operation for removal of retained missiles in the heart.

It would be interesting to know if following this operation, patients develop any discomfort at the level of the diaphragm. Following repairs of extensive gunshot wounds of the diaphragm, patients have complained to me of a peculiar sense of tugging either anteroposteriorly or laterally at the level of the diaphragm. It would be well if all surgeons who

have carried out this operation would get in touch with their patients and determine whether or not any postoperative distress has developed from incision and suturing the diaphragm.

QUESTIONS OF TECHNIC

If on incising the pericardium, the wound in the heart is not bleeding, great care should be taken to clear out the blood from the pericardial sac by suction. The sac must not be irrigated with anything except sterile

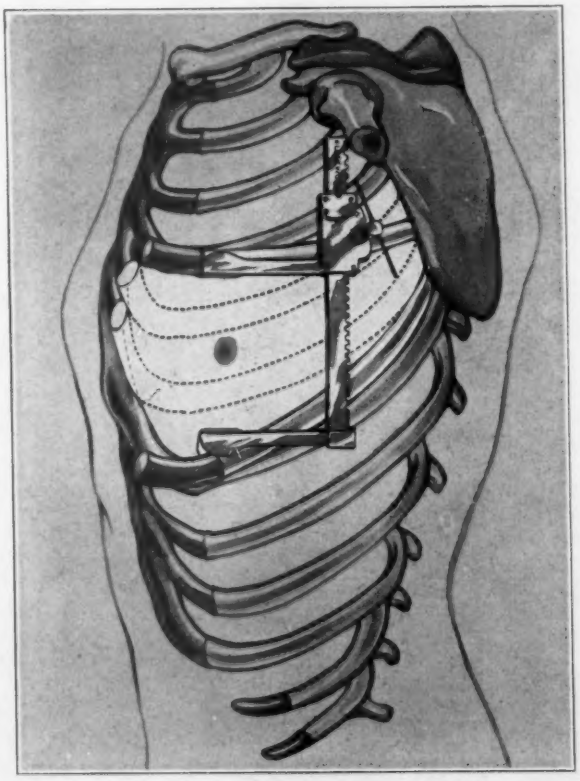

Fig. 10.—Modified Spangaro incision.

water, if at all. Carefully sponge out all clots with moistened cotton pledgets. Take pains to remove all clots from the posterior wall of the pericardial sac, and if possible, from the couch above the transverse sinus. Pneumothorax should be avoided. If it occurs, the hemothorax, if present, should be cleared out and aspiration of the air from the pleural cavity should be practiced after hermetically suturing the pleura. Do not handle the surface of the heart. If required to rotate it, insert a suture through the dependent portion of the apex and rotate or elevate the heart with the suture.

Years ago, Carrel pointed out that the pericardium and heart do not withstand handling as do the pleura and peritoneum. Japanese silk towels or cotton pads covered with Japanese silk should be employed. In an emergency, employ pads of absorbent cotton wrung out of warm water. Gauze, toweling, etc., should not come in contact with the serous surfaces. I never could subscribe to handling manually the heart itself. Except in the rare instances of a laceration of the heart with intense bleeding, it is neither necessary nor judicious to grasp the heart in the hand and deliver it from the pericardium or compress it to

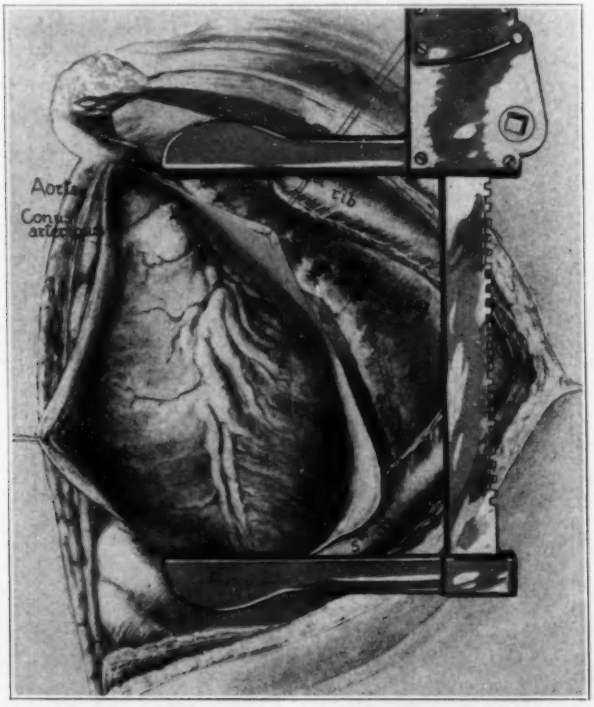

Fig. 11.—Exposure of the heart by intercostal thoracotomy (modified Spangaro incision).

control hemorrhage. Evisceration is dangerous. Constantini, Guibe, Ritthaus, Camus, Dolcetti, Caudemet, Lenormant, Guidone, Kirschner, Tuffier and others have pointed out the danger of evisceration (fig. 14).

To control hemorrhage, digital pressure directly over the wound may be employed or compression of the two vena cava or the ventricles may be gently grasped. After the pericardium has been widely incised and retracted with silk stay sutures and the extent of the injury to the heart has been located and determined, as far as possible, pass a

stay suture through the thickest portion of the apex. With this stay suture as a lever, the heart can be elevated and rotated without handling it manually. Beck's suggestion of digitally controlling the wound with the index finger while steadying the heart with the stay suture in the apex held firmly between the thumb and middle finger of the left hand is a good one (figs. 15 and 16).

Sauerbruch's method of controlling hemorrhage by grasping the base of the heart through the transverse sinus with the left hand is effective,

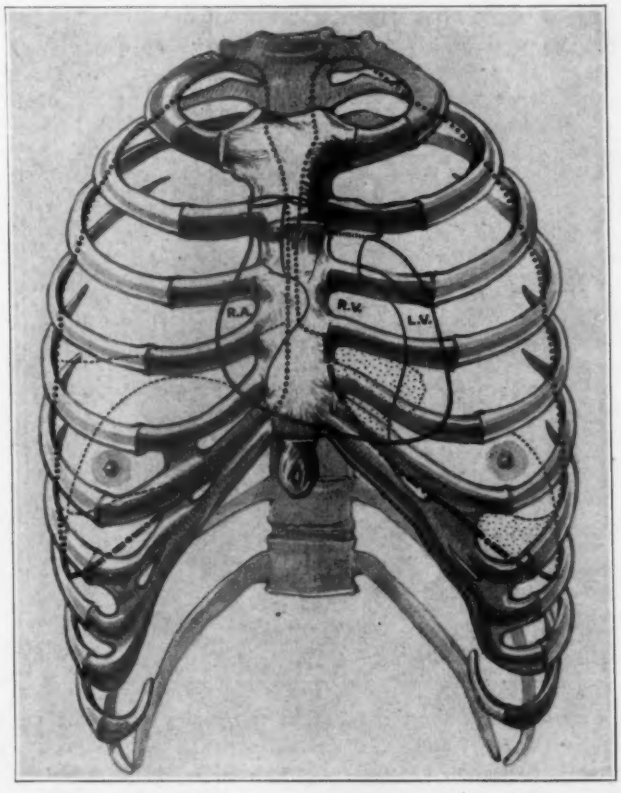

Fig. 12.—The Duval-Barasty median thoraco-abdominal pericardiotomy.

but not usually necessary. Experience in many reported cases had demonstrated that manual compression of the ventricles grasped in the left hand to control bleeding, steady the heart and permit suture of it, is well borne. On the whole, however, any method of manual or digital control is better avoided, if possible. The danger of injury to the lining of the heart, the freeing of blood clot enmeshed in the walls of the cavities, the danger of cerebral ischemia, the incidence of fibrillation and the tendency to traumatize the delicate serous surfaces by com-

pression, with subsequent serous effusion, increasing tendency to infection and development of adhesions, make it necessary that in so far as it is possible and consistent with surgical despatch, manual manipulations of the pericardium and heart should be entirely avoided.

Care in inserting the sutures is necessary or a linear wound may be converted into a jagged, irregular one that cannot easily be closed and then only by a fascial or muscular flap. The suture must not include too much tissue or it produces an anemia of the portion that may slough. It must not be too small or it may cut through.

Silk sutures or twenty day catgut should be employed. Schönborn reported a case that ended fatally, due to the catgut absorbing. The

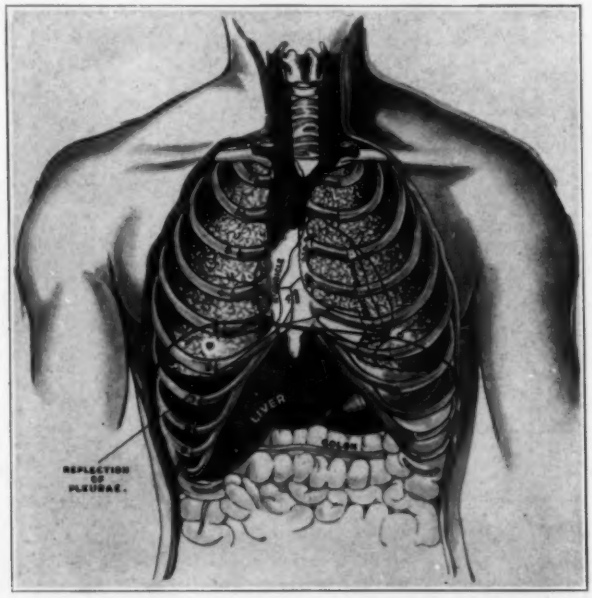

Fig. 13.—Front view of the thorax. The ribs and sternum are represented in relation to the lungs, heart and other internal organs. Note reflection of pleurae with free access to the heart.

auricles are so thin they are difficult to suture and may require to be tied en masse, as advised by Zeidler. In a case in which the muscle tore at the time of suture, Zulehner directly sutured the pericardium to the muscle. Dshanelidze sutured a fragment of fascia lata to the muscle. Jurasz sutured in a plug of muscle.

Tuffier stated that in three quarters of the reported cases of operations on the heart infection had developed, and that the patients were usually cured if the infected parts were opened and drained freely as in purulent pericarditis or empyema.

From experiments on animals, I have long since been impressed with the fact that as delicate a technic for cardiac surgery should be developed as Cushing, that master technician, has developed for brain work.

Experimental work has positively demonstrated that antiseptics, salines, surgical solutions of chlorinated soda (Dakin's solution) or any other type of solution, produce serous effusion with subsequent development of adhesions and should not be employed. The valuable contribution of Cutler and his associates to this question should be

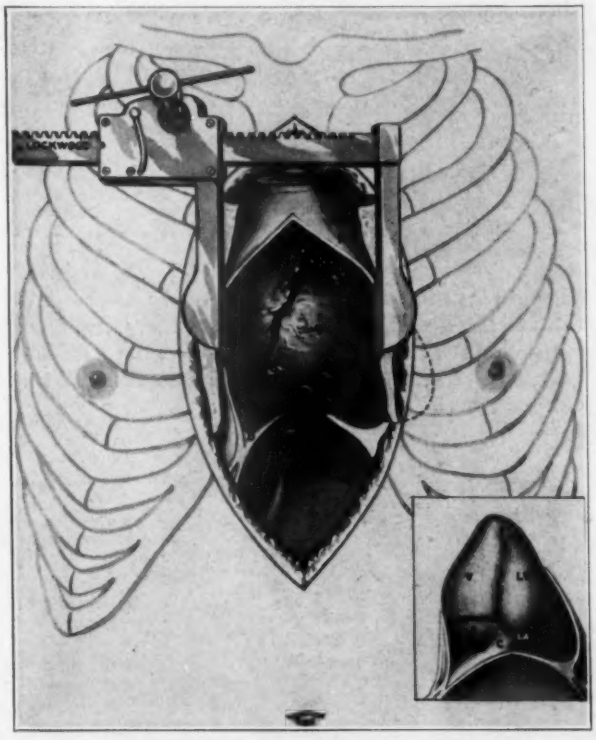

Fig. 14.—Combination of figures from Spalteholz and Merkel's "Anatomy," showing the outlines of the heart, lungs and the pleurae.

carefully read by all surgeons who may be inclined to disregard certain surgical conventionalities.

Deliberately leaving any type of solution in the pericardium or pleural cavity at the end of the operation cannot be too strongly condemned, and need hardly be mentioned before this society.

Curiously enough, blood retained within the pericardium appears to absorb without producing adhesions.

I am opposed to drainage of the pericardium, if hemorrhage has been completely controlled, except in late cases with retained, infected

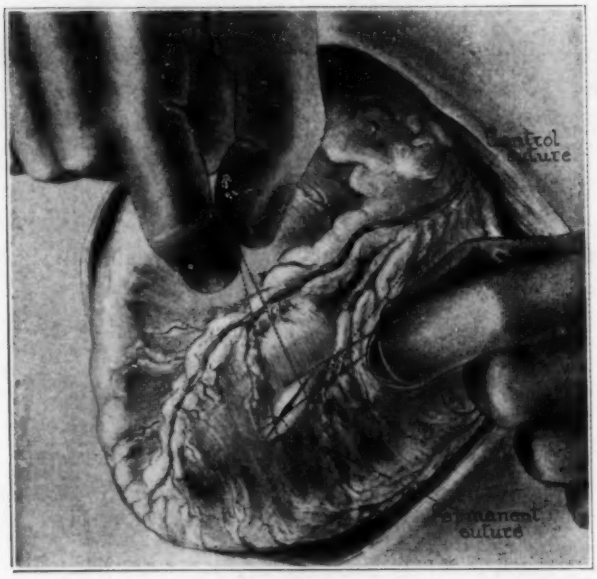

Fig. 15.—The stay sutures are crossed and held by the assistant; hemorrhage can thereby be controlled, and a good exposure of the wound is provided. The permanent sutures can be placed so that adjacent coronary vessels are avoided and a satisfactory approximation of the wound is obtained. The stay sutures are crossed and held under tension. Hemorrhage is thus controlled while the permanent sutures are placed.

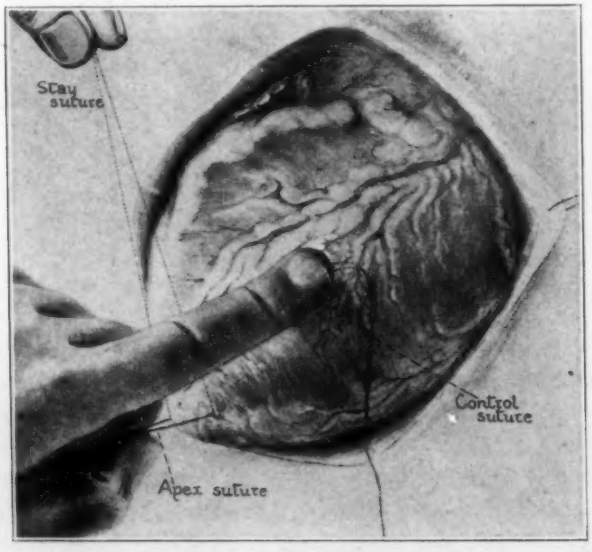

Fig. 16.—Control of hemorrhage from wound preparatory to suture. Traction on apex suture steadies the heart so that the index finger can be maintained effectively on the wound; a control suture is being placed (Beck method).

missiles and infection already established. I would suggest, however, carrying a double silk suture from a dependent portion of the incision of the pericardium, to the surface. This suture will act as a guide for the passing of a curved forcep into the pericardium. If it is not required, on the eighth or tenth day, one end of the suture is pulled out and cut short, and the remnant pulled through. Tuffier advised suturing the pericardium with a Lembert suture, but sufficiently loosely to allow drainage. For a few days a febrile state may develop following operations on the pericardium as on the pleura, but it is of no consequence and, as a rule, will disappear without interference by the tenth day. If fever is maintained, and especially if it gradually gets higher, a purulent pericarditis, mediastinitis or, if the pleura has been opened, an empyema may be developing.

If drainage is required, a Penrose drain placed dependently is best. Suture of the edges of the dependent pericardium to the surface for open drainage is advisable in well established infection.

The dangers and complications of cardiorraphy for wounds of the heart are:

Sudden arrest of the heart.
A jagged irregular wound that cannot be approximated.
Tearing of the wound while being sutured.
Hemorrhage that in a certain percentage of cases can scarcely be checked.
Infection.

The gravest accident during operation is cardiac failure. This should rarely be due to loss of blood with blood donors available. Traction on the heart and evisceration are the main cause of reflex arrest of the heart.

Late postoperative complications are:

An adherent pericardium.
Dilatation of the heart.
Dilatation of the heart and an adherent pericardium.
Extrasystoles and an irritable type of heart with recurring tachycardia.

These late complications have been reported in a surprisingly small percentage of patients.

In 1907, Rhen reported 75 deaths in 125 collected cases. In thirty-three cases, or 40 per cent, death was due to infection; in twenty-eight it was due to hemorrhage.

In 1906, Borchardt collected seventy-eight cases in which operation had been performed with thirty-nine deaths, twenty-one of which were due to infection.

In the 150 collected cases of suture of the heart reported by Vaughan in 1909, ninety-eight deaths occurred. Forty-four of these patients lived at least twenty hours after operation, and died mainly of infection.

Guibal reported forty cases with eighteen deaths due to infection.

Constantini, in 146 deaths, pointed out that thirty-nine occurred immediately and forty-two in thirty-six hours after operation due to hemorrhage, shock or reflex arrest of the heart, but mainly due to direct loss of blood.

Tuffier stated that the average mortality following the operative treatment of wounds of the heart is 50 per cent and is approximately 5 per cent higher for stab wounds than for gunshot wounds; also, that approximately 2 per cent of deaths is due to pulmonary embolus.

MORTALITY STATISTICS

A high percentage of gunshot wounds are rapidly fatal, especially with high velocity trachectory such as employed in modern war. Most of the patients who have survived gunshot wounds of the heart have probably been hit by spent or ricochet bullets or from pistols of small caliber and low trachectory.

In most patients who come to operation with wounds of the heart, the wound involves the anterior rather than the posterior aspect of the organ.

The majority of stab wounds are amenable to surgical treatment, if promptly dealt with. Probably as Smith has pointed out, patients with heart wounds have three chances in four of surviving long enough to permit surgical intervention; one chance in ten of healing spontaneously and a little better than one chance in two of being cured by surgical treatment. Lund pointed out that approximately 80 per cent of patients with heart wounds should be operated on and half of them should recover. Cardiorrhaphy has raised the percentage of cures from 10 to 40 per cent.

Constantini reported twenty-seven patients with wounds at the base of the heart with nineteen deaths (70 per cent) and thirteen patients with an associated cardiac and abdominal wound with ten deaths (77 per cent).

Ballance, in his Bradshaw lecture, collected 152 cases of wounds of the heart with a mortality rate of 31.57 per cent. Sixty-three of these occurred during the war with sixteen deaths, or 26.03 per cent.

Tuffier reported 305 operations with 149 deaths, and 5 cases in which the final result was not clear—a mortality rate of 49.66 per cent.

Smith collected fifty-eight cases since Pool's paper in 1912 with a mortality rate of 33.33 per cent. Only 25 per cent of patients that have survived operation have been free from infection.

If the lessons learned from the development of thoracic surgery during the Great War are applied—active resuscitory measures, heat, blood transfusion, etc., and if operation under a select anesthesia is under-

taken within from one to four hours of receiving the wound, at least 60 per cent of all wounds of the heart should be amenable to surgical treatment.

CHOICE OF ANESTHETIC

Local anesthetic associated, if necessary, with a paravertebral block and gas-oxygen analgesia, if the patient becomes restless or the manipulations within the thorax excite a troublesome cough, is the anesthetic of choice.

After the pericardium has been incised, thereby relieving alarming pressure on the auricles and the bleeding from the heart itself has been

TABLE 9.—*Mortality Table*

		Number of Patients	Operated	Not Operated	Gunshot Wounds	Stab	Interval Till Death	Infection	Hemorrhage	Number of Deaths, per Cent	Number of Recoveries, per Cent
Fischer.....	1868	452	...	..	Both		..	..	..	90	10
Rehn.....	1896	124	124	..	Both		..	..	..	60	40
Borchart.....	1906	78	78	..	Both		..	21 deaths	..	50	50
Salomoni.....	1901	97	..	..	Missiles	..	..	..	..	51	40
	1906	160	..	..	in heart	..	..	..	..	38	62
Vaughan.....	1909	150	..	..	Both		44 lived 24 hours	44	..	65	35
Peck.....	1909	160	..	..	Both		..	..	..	63.8	36.2
Pool.....	1912	452	..	..	72	304	104 a few minutes 219 later	..	..	84.1	15.86
Ballance.....	....	58	..	..	..	..	..	..	..	24.2	75.8
Constantini...	1920	287	287	..	Both		..	..	38	50.88	49.12
Tuffier.....	1920	305	305	..	Both		..	..	..	49.6	50.4
Smith.....	1923	58	..	..	Both		..	..	..	33.4	66.6
Klose.....	1923	57	32	24	57	..	..	..	..	31.4 37.1	68.6 62.8
Dshanelidze...	1924	535	..	..	132 or missiles	402	..	..	..	66	34
Lenormant...	1921, 1925, 1926	3	3	..	..	3	..	..	..	66.6	33.3
Lockwood.....	1928	51	48	3	35	16	..	..	..	23.2	76.8

controlled, the operation under such an anesthetic may be carried on in a more deliberate and protracted manner without risk to the patient from the anesthetic per se.

A considerable percentage of patients with serious wounds of the heart are in profound shock or a state of semi-unconsciousness from hemorrhage and require practically no anesthesia for the initial incision. A certain percentage of patients have intense respiratory distress and precordial pain. They are restless, tossing about like mad men. Such patients have a serious lesion, distress due to hemorrhage with pressure, and require immediate operation. The "mass ether" anesthesia is ideal for such cases. In less than one minute the patient is completely

relaxed, and examination of the wound, cleansing of the skin and preparation for operation may be carried on. When the field of operation has been properly prepared, local anesthesia may then be employed and the operation continued, the anesthetist maintaining a light gas-oxygen analgesia, if a machine is available. This "knock-out ether" anesthesia is not at all objectionable to the patient. It is so instantaneous that they have no distress. I have taken it three times myself to try it and can personally bear evidence that it is not at all distressing. It acts exactly and as quickly as a knock-out blow. It is free from danger. During the Great War it was used in literally hundreds of cases without mishap. If a prolonged ether anesthesia is required, the anesthetist merely carries on by his own pet method. Delay in inducing anesthesia in distress due to hertz tamponade may be fatal.

CONCLUSIONS

1. The heart is tolerant of severe trauma and manipulation.

2. The danger of hertz tamponade must be constantly in mind. One must appreciate that if the patient has lost much blood, with the lowered tension, the shock and the ability of the heart to withstand trauma, the wound in the heart may temporarily cease to bleed. As the patient's condition improves, and the blood pressure rises, delayed primary hemorrhage may occur and the patient be found "in extremis" again if not constantly observed.

3. Injury of the bundle of His is often fatal. Pressure on the bundle of His or kinking and rotation of the heart by interfering with conduction through the bundle in doing a cardiorrhaphy, may cause the heart to fibrillate or arrest entirely.

4. Wounds of the heart heal by the formation of a cicatricial scar, not by true regeneration of muscle. Spontaneous rupture of the scar has frequently occurred following strain.

5. The coronary arteries may be ligated without grave sequelae if the ligature is applied distal to the bifurcation; when applied proximally, the results are fatal.

6. Digital control of bleeding from a laceration of the heart should be resorted to if necessary. Forceps should not be applied.

7. No. O chromic catgut sutures or black silk carried on fine non-cutting needles are highly satisfactory for closure of wounds of the heart. Sutures should not be carried into the cavity of the heart. Care should be taken in inserting sutures and tying them to prevent cutting.

8. The pericardium should be closed if one is reasonably sure of the asepsis. If drainage of the pericardium is necessary for sepsis, it should, if at all possible, be dependent. The pleura should be hermetically sealed finally if opened.

9. A supporting stay suture through the apex is most valuable if elevation or rotation of the heart is necessary.

10. Gauze should not be employed within the pericardium.

11. Sterile water only should be employed to cleanse the pericardium. No antiseptics should be applied to the serous surfaces.

12. Great care must be taken to avoid infection, bearing constantly in mind that most of the deaths after twenty hours have been due to infection.

13. A select anesthesia should be employed.

14. The great value of cardiac massage to overcome sudden arrest of the heart should not be forgotten.

Cushing has spoken of cardiac surgery as the "Last Great West of Surgery." The pioneer work has been well done. The future is bright. Medical problems of the heart hitherto incurable are about to yield to surgical measures. Pericardotomy for purulent pericarditis should be more frequently employed, and should be undertaken early in the disease.

Cardiolysis in selected cases should be more commonly practiced.

Cardiorrhaphy for injuries of the heart should be promptly undertaken when indicated, especially if hemorrhage is occurring.

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REMOVAL OF BULLET FROM THE PERICARDIUM UNDER LOCAL ANESTHETIC

REPORT OF A CASE

T. C. DAVISON, M.D.
ATLANTA, GA.

My reason for reporting this case is that it is one of more than usual interest. The thorax was penetrated by two bullets, either of which might have caused death. The first perforated the lung near the hilum and evidently injured one of the larger blood vessels, resulting in a massive hemothorax, pneumonia, and later, in empyema. The other injured the pericardium, resulting in pericarditis, and finally, in a localized abscess of the pericardium. The patient suffered from primary shock, internal hemorrhage and infection, but finally recovered.

REPORT OF A CASE

History.—A man, aged 27, was admitted to the hospital on March 30, 1923, with two penetrating gunshot wounds of the left side of the chest; one had its point of entrance in the third interspace in the nipple line; the other, in the fifth interspace and slightly to the inner side of the nipple line. There were no wounds of exit. Powder burns were present on the skin around the wounds. The patient was in a state of collapse or shock when admitted to the hospital. The blood pressure was: systolic, 88, and diastolic, 68; the pulse pressure, 20.

Examination.—On physical examination, the right side of the chest was normal; the left side of the chest was hyperresonant anteriorly and dull posteriorly to the midaxillary line. The heart sounds were rapid, indistinct and muffled. The area of cardiac dulness was increased to the right and left and downward. The first x-ray picture showed that the entire left side of the chest was cloudy, due to the hemothorax, and only one bullet was visible at the level of the tenth rib posteriorly and evidently free in the pleural cavity. The patient was given morphine freely; the left side of the chest was strapped, and he was treated for shock and internal hemorrhage. He reacted slowly and was seriously ill for several weeks.

This case might be classified as a mental case, the wounds being self-inflicted with suicidal intent; therefore, the patient did not cooperate, but was antagonistic and hard to control, which added to the difficulties. His most prominent symptoms were dyspnea, pain in the left side of the chest, hiccup, restlessness, muscular twitching, cough, cyanosis and at times delirium. The temperature ranged from normal to 102.6 F.; the pulse rate, from 100 to 136, and respirations, from 12 to 36, according to the quantity of morphine administered. Thirty-six hours after he was admitted to the hospital the blood count was: red blood cells, 2,530,000; hemoglobin, 50 per cent; white blood cells, 13,900; polymorphonuclear leukocytes, 97 per cent. On the second day, there were fine moist râles in the apex of the left lung and a pericardial friction rub.

The patient evidently developed a traumatic pneumonia in the apex of the left lung as a result of the perforating wound produced by the upper bullet, and a pericarditis, produced by the impact and presence of the lower bullet. At the end of a week aspiration was performed on the left side of the chest and several hundred

cubic centimeters of blood was removed. Five days later, 2,000 cc. of blood was aspirated; a week later, 1,250 cc. was removed, and at intervals of about a week smaller amounts were aspirated until altogether a total of more than 5,000 cc. had been removed. The first specimen was sterile; later, staphylococci were found, and at the last, bloody pus was removed. As the pressure in the left side of the chest was relieved by aspiration, the precordial pain became more pronounced and annoying. The temperature continued to run a septic curve. There were septic sweats, and the patient complained of pain in the left shoulder and arm; he developed acute arthritis in one wrist, one ankle and the fingers of both hands. Blood cultures were repeatedly negative. He continued to hiccup at intervals.

Subsequent x-ray pictures showed one bullet directly over the heart shadow, and on fluoroscopic examination, it appeared to be either in the pericardial sac or embedded in the heart muscle as it moved simultaneously with each heart beat. A lateral view showed that it was anterior to the heart, but in direct contact. The pericarditis was still present; the pulse rate ranged from 96 to 120, and the precordial pain was present almost constantly. It was decided to remove the bullet from the pericardium first, and later the one in the pleural cavity.

Operation.—On May 8, or five weeks after he was shot, he was operated on and the bullet removed. He was given a preliminary hypodermic injection of morphine and scopolamine, and then the tissues were infiltrated with 2 ounces (59.2 cc.) of 1 per cent procaine hydrochloride. An incision was made over the third rib at its junction with the sternum, and a portion of the third and fourth costal cartilages was removed. The pericardium was exposed and incised and the edges retracted. The cavity and the heart were inspected and palpated carefully and thoroughly. The bullet could not be located either in the pericardial sac or in the heart muscle. Each time the heart was palpated it became rapid and irregular, but it would quiet down when the examining finger was removed. When the palpating finger was turned from the heart, a thickened and bulging area was found to the left of the incision in the sac. This mass was aspirated and found to contain pus. The pericardium was closed with no. 0 chromic catgut sutures, and then the abscess in the pericardial wall was opened, 4 cc. of pus evacuated and a no. 25 caliber brass-jacketed bullet removed. A small rubber tissue drain was placed in the cavity and the skin closed.

The patient's condition was good. He was nervous and complained of some dyspnea. His pulse rate was 148 and the rate of respirations, 44. Three hours later, there was considerable hemorrhage from the wound, which was controlled by pressure. I think it came from one of the intercostal vessels. The drain was removed on the fourth day, and the wound healed gradually.

The patient's general condition improved rapidly but he had also an empyema. Two weeks following the first operation, a portion of the tenth rib was resected posteriorly, under procaine hydrochloride, and the remaining bullet was removed from the pleural cavity; also about 500 cc. of bloody pus was evacuated. A tube was inserted and the wound closed around it. A surgical solution of chlorinated soda (Dakin's solution) was used daily to irrigate the empyema cavity. The patient convalesced rapidly and was finally dismissed from the hospital on the ninety-fourth day, entirely well.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. WHITE, HEDBLOM, OCHSNER AND HERNMAN, SHIPLEY AND HORINE, BECK AND CUTLER, LOCKWOOD AND DAVIDSON

DR. WILLY MEYER, New York: I cannot speak of operations on the heart proper, and I have not had an extensive experience in cases of pericarditis which

require operation. At the same time, I think a symposium like this ought to be discussed. The little I can contribute, I am glad to offer.

In general, I can only repeat what I said at the last meeting in New York when papers on pericarditis were on the program. Personally, I believe that closed drainage is the best for the physiologically closed cavities of the chest, the pleura and the pericardium. I am not speaking of children. Last year, Dr. Peterson told us of a number of interesting and successful cases in which the sac was left open and a tube was not introduced. Of course, similar results might be obtained in adults by the same procedure. Judging from the cases of acute suppurative pericarditis of adults that I have seen, open drainage (with the help of tubes) seems to be unsatisfactory. It appears best to me that the treatment of the suppurating pericardium should not be much different from that of the suppurating pleura. Both sacs are situated within the chest, both are subject to the effects of negative pressure, as long as the chest has remained or been made airtight, as well as to positive pressure after the incision has been made and drained.

From what I could observe in those cases of acute empyema in which operation was performed at the Lenox Hill Hospital in New York, I would conclude that what we ventured to call "the physiologic treatment of empyema of the pleura" is the best for the patient. With the patient under local anesthesia, we make a resection of a piece of rib, long enough so that one may eventually put in the hand and clear the pleural cavity completely of its contents as far as coagulation of fibrin is concerned, break up adhesions, etc. Then after the cavity has been washed, or not washed, according to the inclination of the surgeon, a second small incision is made, somewhat posteriorly, two intercostal spaces lower, for airtight drainage. The main incision is then closed by layer sutures. In almost every case we have seen primary union of this wound. After a few days that part of the lung which is not infiltrated any longer has distended, because it is under physiologic conditions, and has become adherent to the costal pleura.

The old Buelau method of closed drainage is also excellent and will save the life of many patients who could not stand a more radical operation. However, it does not permit one to cleanse the pleural cavity of the coagulated fibrin and to examine the pleura and lung by direct inspection.

Now I would not say that the pericardium is exactly on the same basis as the pleural cavity, but I believe it is on a similar one. I can never forget—and I said so last year—that wonderful case of Dr. Whittemore, of a greatly emaciated boy with suppurative pericarditis who was in a desperate condition. Dr. Whittemore did not dare to do a radical operation, but used Buelau's method of closed pleural drainage for the distended pericardial sac, and saved the patient's life. Of course, nobody can prove that he would have failed had he made an incision, but certainly Buelau's method was the mildest and proved successful.

Unfortunately, I no longer have the chance of operating on many of these patients, having passed the age limit. We older men here are in a rather peculiar situation. We can do the talking and give our impressions, but we cannot prove our statements by reporting a number of cases in which our treatment was used. It is a rather difficult experience. But we do not complain, for the younger generation has its rights, and we are satisfied that we have made room for it and for the following generation.

I repeat, from all I have seen, heard and read, but without being able to corroborate my statement with a series of patients successfully operated on, I believe that the rules governing the pleura hold good for the pericardium. If I had such a patient today, I would do the ordinary pericardiotomy and clear the sac of its contents, also the posterior space where sometimes coagulated fibrin is present. I

would clean it out, if necessary wash it out, and, under the guidance of my eyes, make a careful examination of the cavity, as we do in the pleura. Then I would make a buttonhole, sideways in the pericardial sac, just large enough to allow the drainage tube to pass, and close the original incision of pericardium and wall of the chest completely. The end of the tube dips into the fluid of the syphon bottle under the bed. I believe that this method of treatment would give good results, and that the mortality in suppurative pericarditis in adults would be less than it is now with the use of the open tube drainage. Dr. Cutler and his associates have done difficult, ingenious and persistent work which will meet with lasting success in the end.

Dr. Howard Lilienthal, New York: I want to say, in the first place, that I think Dr. Cutler should be given a great deal of encouragement for the work he has done. I think he ought to keep on with it, for he is still only at the beginning. I would suggest that if he wants to use a straight instrument in getting into the auricle, it could be done by making a wide intercostal thoracotomy, putting in a rib spreader and reaching the heart through the pericardium. I have made this exposure in searching for bullets, and there has always been plenty of room.

I have had three cases of suppurative pericarditis. One little girl died; the other two patients recovered. One case was followed up for twenty-two years, and there was no adhesion whatever between the external part of the chest and the heart. The x-ray pictures showed a wide, fully normal space between the two structures, and no tube was used. The method of suturing the pericardium to the skin was employed. I unfortunately fail to remember the name of its originator. I believe that the danger of adhesions, if you employ rubber tubes, is considerable. The constant motion of the heart acts as a massage and tends to prevent the formation of adhesions and to assist the outflow of the pus.

Dr. R. B. Bettman, Chicago: I thought it might be of interest to the society to hear the report of some work I am doing now in regard to the transposition of the heart. About two months ago our cardiologist asked me whether he might take electrocardiograms in all of my cases of empyema and artificial pneumothorax, before and after the operation. We have been doing this since that time. It is of interest to note that the electrocardiograms have not varied appreciably, although in both the cases of empyema and the artificial pneumothorax the position of the heart has been shifted greatly. The only significant fact, therefore, thus far revealed is that the mediastinal structure shifts as a whole, but that the heart itself does not rotate. If the heart had rotated, the electrocardiogram should have differed. As I say, we have been doing this work for only a short time, and therefore do not want to draw any conclusions but give this information for what it is worth: in pressure operations of the chest such as the removal of fluid or the injection of air there is no rotation of the electric axis.

Dr. E. W. Peterson, New York: I have not had any experience in the actual surgical measures on the heart, but at our meeting a year ago, I reported three cases of pericardiotomy for purulent pericarditis with two recoveries. In my first case, I used irrigation and drainage of the pericardial sac, and the patient died promptly. In my next two cases, the pus was evacuated by aspiration, and no drainage material was placed in the pericardium. The patients were instructed to lie face downward, for a few minutes, every two or three hours, thus securing gravity drainage. The wounds were not irrigated.

In the matter of physiology and physics, the pericardium differs essentially from the pleural cavity. Closed drainage may be used with advantage in pleural suppuration, but I can see no special reason for using closed drainage in the pericardial cases.

DR. EVARTS A. GRAHAM, St. Louis: I think that this series of papers represents not only excellent experimental work, but careful clinical observations, and is a masterful expression of honesty and candor.

I would like to say, however, a little more particularly, that I have been interested in what Dr. Bettman said a moment ago about the failure of the electrocardiogram to show any shifting of the position of the heart, because this coincides exactly with some results, as yet unpublished, that have been obtained by Dr. Duff Allen, working with us.

Another point I would like to refer to is the point brought out by Dr. Hedblom about massive atelectasis being associated with carcinoma of the lung. It would seem to me that in all probability, as was so well demonstrated yesterday, the occurrence of dextrocardia in these cases was probably due to the bronchial obstruction with atelectasis, with the result that the structures were pulled over. The dextrocardia, therefore, probably had no particular specific relationship to the carcinoma. I have no doubt that was in Dr. Hedblom's mind, but I thought some mention ought to be made of it as he might want to speak of it again.

DR. CARL A. HEDBLOM, Chicago: I have only two points to add. One is with reference to the question of atelectasis producing dextrocardia in cases of malignant disease of the right lung. In two of the three patients whom I treated there was no doubt but that the atelectasis was responsible for the distraction of the mediastinum structures to the right. In the third patient, however, there was an extensive fibrosis associated with the carcinoma, and the bronchi were not obstructed, as a postmortem examination showed, so I think we must recognize that atelectasis is not the reason, necessarily, for the shifting even in cases of carcinoma.

With reference to the electrocardiogram, unfortunately, in the cases I have observed, the reading was not taken, but in studying the literature the impression I gathered is that in all cases of acquired dextrocardia, regardless of whether the heart has rotated or not, the electrocardiographic reading is that of the normal heart, while in cases of congenital transposition of the heart there are characteristic deviations. I am not in a position to feel very certain of my ground there, but that is the impression I get from a review of the literature.

DR. POL N. CORYLLOS, New York: I have had the opportunity of performing two operations along these lines. The only special feature in the first was that I was obliged to use a high spinal anesthesia, because I was afraid to use local infiltration. It was a real operative success, and I feel the same way as Dr. Graham that here, perhaps, we act more by decompression than in any other way.

So far as the experimental work on pericarditis by Dr. Oschner is concerned, I have had the opportunity of experimenting on the heart to produce mitral stenosis with radium seeds in the beginning, and later with radium bulbs. I had a high mortality in the dogs at first and was afraid that postoperative dilatation was the cause, especially since I used to suture the pericardium completely at that time, and I had the idea that perhaps the confinement of the heart within the pericardium was the cause of the circulatory trouble and postoperative death. When I dissected the pericardium completely, I had much better results, and in a case of a dog with completely resected pericardium that I killed nineteen months later, I was surprised not to find adhesions between the heart muscle and the lung.

I feel as the speakers do that it will be necessary to try by all means at our command to produce an artificial mitral stenosis; I think, judging by the studies I am now making, it will perhaps be possible by using very weak bulbs of radium emanation.

Dr. Elliott Carr Cutler, Cleveland: It was a mental and spiritual ordeal to present to any society such figures as we presented this morning. However, we felt that, even if we abstained from further attempts, others who in years to come might attack this problem afresh should have all the benefit of our mistakes and discouragements.

We seem to have learned something from both our experiments and the clinical attempts, and the discussion here has added valuable points. Dr. Graham spoke about the possible advantages simply from the thoracic decompression. This matter had already been called to our attention by Dr. Henry Christian. This possibility alone merits further study.

Dr. Coryllos some time ago attempted to produce mitral stenosis by radium, and some of his results were published. We have attempted similar experiments, but neither by this method nor by any other of the many methods attempted have we been able to reproduce a valvular lesion similar to that which occurs in man. Could such a condition be produced, such figures and disasters as we have reported today would not exist.

Dr. A. L. Lockwood, Toronto: I want to correct an impression that one of the members advised me I had left. I was not criticizing Trendelenberg for operating on twelve of his patients with pulmonary embolus. The two patients whom I prepared for operation and who recovered spontaneously had had repeated emboli, and because of the fact that they had not died during the first attack we had everything ready to undertake surgical measures should they have what would appear to be a fatal attack. Before we found it necessary to undertake what might be a fatal operation, they recovered.

I would like to make a plea that in all centers the surgeons should have in mind some definite means by which to do a cardiorrhaphy when indicated, the procedure to be undertaken; and that they should be prepared to undertake the operation hurriedly.

PULMONARY ABSCESS AND PULMONARY GANGRENE

CLINICAL COURSE AND PATHOLOGY *

B. S. KLINE, M.D.
AND
S. S. BERGER, M.D.
CLEVELAND

The lesion in many cases of so-called typical abscess of the lung has been found to be gangrene. Instead of a grayish area of suppuration without appreciable odor, the lesion is ragged, brownish or greenish and penetratingly foul-smelling. The sputum in these cases is foul smelling, grayish brown or grayish green and contains characteristic oral spirochetes, fusiform bacilli and vibrios. In cases of abscess, on the other hand, it is whitish yellow, mucopurulent or purulent, without appreciable odor and contains pyogenic organisms, usually staphylococci.

As patients with pulmonary gangrene do not respond well to the treatment for abscess but are frequently cured by arsphenamine therapy,[1] the differentiation between these two diseases is imperative.

Anatomically, the pulmonary abscesses observed were of two types:

1. The embolic type of abscess, which involves a number of lobes, is multiple and relatively small, is associated with areas of suppuration elsewhere in the body, and represents a manifestation of pyemia or septicemia.

2. The aspiratory type, which is usually limited to one lobe (a lower lobe most frequently), is not infrequently multiple and at times is confluent and associated with a regional pneumonia but not with a pyemia or a septicemia. The abscesses varied in size, were reddish gray or grayish yellow and did not have any appreciable odor. In a few early cases, the lesions were reddish. Smears and cultures showed staphylococci in all. Sections of early lesions stained by the Gram method showed numerous clusters of staphylococci in the bronchial branches and in the alveoli, whereas the regional blood vessels were uninvolved.

The majority of observers and investigators believe that the organisms producing the so-called typical abscess of the lung are aspirated from the oral cavity. A number, however, believe that this lesion, especially when it follows operation, is produced by organisms which

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* From Mount Sinai Hospital.

1. Kline, B. S., and Berger, S. S.: Spirochetal Pulmonary Gangrene Treated with Arsphenamines, J. A. M. A. **85**:1452 (Nov. 7) 1925.

reach the lung through the blood stream. A recent article [2] contains the following review of the subject:

The clinical and experimental evidence for embolism is well presented in the excellent communications of Cutler, Schlueter, Weidlein and Holman and of Fetterolf and Fox. This evidence as presented in a recent communication by Schlueter and Weidlein is as follows:

Our belief that postoperative lung abscess results from embolism, a mechanism produced by the dislodgment of an infected thrombus from the vessels of the operative area, is based on the following facts:

1. The definitely proved existence of the condition of fatal postoperative pulmonary embolism. This supposes the possible scattering from any wound of single or multiple emboli into the venous circulation.

2. The frequent development of lung abscess after operations performed in infected or potentially infected fields. In this class we refer particularly to nose and throat operations, especially tonsillectomy, and to operations performed on the gastro-intestinal tract.

3. The high percentage of occurrence after operations performed in mobile operative areas. Thrombi are easily dislodged from such regions as the pharynx and epigastrium. In operations on the brain in which the skull acts as a splint the percentage of postoperative pulmonary complications is almost nil.

4. The not uncommon appearance after operations in which local anesthesia is employed.

5. The failure to prevent postoperative pulmonary complications with the constantly improved methods of giving inhalation anesthesia.

6. The greater frequency of lower lobe involvement. This is explained by the greater volume of blood and the more direct course of the pulmonary artery to these lobes.

7. The often symptom-free period following the operation before the onset of the complication. If the aspiration mechanism were the causative factor, the appearance of the symptom would be early.

8. The sudden pain in the chest that frequently constitutes the initial symptom and the often severe and stormy associated clinical course that follows before rupture and evacuation occur.

9. The acknowledgment by bronchoscopists that typical lung abscess is rare with the lodgment of foreign bodies even deep in the air passages.

10. The unsuccessful attempts at experimental production in animals by the introduction of infected materials by way of the air passages, either by transtracheal implantation or by aspiration.

11. The comparative ease with which lung abscess can be produced by the intravenous injection of infected materials.

COMMENT ON EMBOLIC THEORY OF ABSCESS OF THE LUNG

Although pulmonary abscess was produced in dogs by the intravenous injection of a large embolus containing staphylococci, streptococci, pneumococci and colon bacilli, the process eventuated in healing and not in a progressively enlarging

2. Kline, B. S.: Pulmonary Abscess and Pulmonary Gangrene, J. A. M. A. 90:2008 (June 23) 1928.

lesion. That such a progressive lesion was not produced is probably due more to the organisms employed than to the route. The experimental abscesses reported by these investigators are similar to the embolic pulmonary abscesses observed clinically in cases of septicemia and pyemia. In cases of this type in man observed at autopsy, the abscesses were invariably multiple, involving several lobes and varying in diameter from several millimeters to about 1.5 cm. In one case they were confluent in places. The gross lesions were grayish or reddish gray without appreciable odor. Many were just below the pleura, and this structure was frequently involved. Microscopic examination of sections stained by the Gram method showed clusters of staphylococci first within the lumen of a blood vessel, then within the walls, the lumen at this time usually containing a thrombus. Apparently following the inflammatory process in the walls of the vessel there was a spread of the staphylococci and of the suppurative process into the regional lung tissue.

Although of interest in connection with the evolution of embolic abscess already described, the experiments of Cutler and his associates in our opinion do not throw any light on the pathogenesis of the so-called typical lung abscess of man. The following facts are more convincing evidence that in these cases the organisms reach the lung by aspiration:

1. The frequent occurrence of aspiration of foreign material is borne out by the finding at autopsy of deposits of coal pigment in the lungs of all adults.

2. Pneumonia undoubtedly following the aspiration of food particles and bacteria during the unconsciousness of coma or of anesthesia is an occasional autopsy observation. On microscopic examination, sections from these cases show the foreign material and bacteria in the bronchial branches and in the alveoli, surrounded by inflammatory exudate.

3. It was reported by Myerson that bronchoscopic observation immediately following tonsillectomy under general anesthesia showed the presence of blood and mucus in the bronchial tree in 155 of 200 cases. Myerson concluded that the failure of evacuation of infected material is the most important factor in the causation of pulmonary abscess. Among the reasons given for this failure are the loss of action of the cilia, the lessened elasticity and compressibility of the lung and a local immobility. Furthermore, it has been shown experimentally that rabbits receiving considerable numbers of pneumococci in the trachea just beyond the larynx get rid of them without suffering appreciable involvement of the lungs, whereas in those animals in which the same number of similar organisms has been introduced into the air sacs an inflammatory process invariably develops.

4. The production of pneumonia by intrabronchial inoculation of pneumococci in dogs by Meltzer and Lamar, in rabbits by Winternitz and Hirschfelder, and by intratracheal inoculations in monkeys by Cecil and Blake, proved that aspiration can explain the manner in which organisms reach the lung in man.

5. There is evidence for the belief that the various inflammatory lesions of the lung may be brought about by the aspiration of the causative bacteria from the mouth during the deep sleep following fatigue. In much the same way ether anesthesia increases the opportunity for aspiration into the lung and at the same time renders the body incapable of expelling the foreign material.

6. The occurrence of severe pulmonary inflammation containing innumerable bacteria following clean operations in clean fields on patients under general anesthesia indicates that in these cases the bacteria are undoubtedly aspirated from the oral cavity. Likewise, in clean cases done under local anesthesia, the bacteria most certainly reach the lung by aspiration and not by embolism.

7. An anatomic study, including examination of Gram and Warthin-Starry stained sections of early lesions of pulmonary abscess and of pulmonary gangrene, reveals that the process in these cases is one of inflammation starting in and about smaller bronchial branches. This is quickly followed, however, by changes characteristic of abscess when staphylococci or other pyogenic organisms predominate in the lesion, and more slowly by changes characteristic of gangrene when spirochetes, fusiform bacilli and vibrios of oral type predominate. The spirochetes are present not only in the areas of necrosis but also at the advancing periphery.

8. The experimental production in a rabbit of pulmonary gangrene by the intrabronchial injection of material from a carious tooth containing innumerable spirochetes and fusiform bacilli is proof that aspiration of these organisms may produce pulmonary gangrene. This experiment is confirmed by those of Smith, who reported the production of experimental aspiratory abscess in mice, guinea-pigs and rabbits by the intratracheal inoculation of material from about the teeth of patients with moderately severe pyorrhea, containing spirochetes, fusiform bacilli and vibrios. More recently, Crowe and Scarff, and Allen report the production of lung abscess in dogs by the intrabronchial inoculation of material containing oral spirochetes.

Schlueter and Weidlein state that "in a census of recent writers forty declare themselves in favor of aspiration while only ten favor embolism as the direct cause of lung abscess." From the available evidence, the view of the majority is apparently the correct one.

If it is true that the organisms producing pulmonary abscess and pulmonary gangrene are aspirated from the oral cavity, the reduction of trauma by the surgeon in the operative cases as advocated by Cutler will do less to prevent these conditions than proper oral hygienic and therapeutic measures. Such a program of preoperative oral care is now being followed at Mount Sinai Hospital of Cleveland.

In contrast to abscess, the fully developed characteristic lesion of gangrene is ragged, penetratingly foul-smelling, brownish or greenish, and smears and sections from it, when properly stained, show innumerable spirochetes, fusiform bacilli and vibrios. The sputum in these cases is foul smelling, grayish brown or grayish green, and when carefully washed and stained by strong carbol fuchsin solution or by the Fontana method show the characteristic spirochetes, fusiform bacilli and vibrios.

In the majority of cases observed clinically, the pleura was involved, frequently so severely as to require thoracostomy; in two cases following repeated thoracentesis, gangrene of the wall of the chest developed. One case of this series was associated with active pulmonary tuberculosis.

In the past five years at this hospital, pulmonary gangrene has been observed three times as frequently as pulmonary abscess. This incidence of pulmonary gangrene, although strikingly high, is not generally known. Graham,[3] however, in discussing the treatment for pulmonary suppuration last year, stated:

Many of those cases associated with spirochetal infections become healed in a very dramatic manner merely by the administration of an intensive course of treat-

3. Graham, E. A.: The Treatment of Pulmonary Suppuration, Ann. Surg. 86:174 (Aug.) 1927.

ment with neosalvarsan. It is becoming more and more evident that such cases are much more frequent than was formerly supposed. In the experience of Singer and myself during the last year, as many as 74 per cent of our cases were found by Dr. Varney of the department of bacteriology to be associated with large numbers of fusiform bacilli or spirochetes in the sputum.

As not more than 150 cases of pulmonary spirochetosis have thus far been reported in the United States,[4] it is highly probable that many of the cases diagnosed as pulmonary abscess from clinical and roentgen evidence but not supplemented by a study of the sputum as outlined, were, in reality, cases of pulmonary gangrene.

The spirochetes, fusiform bacilli and vibrios of pulmonary gangrene are identical with those present in practically all adults in the interproximal spaces between the gums and teeth.[5] The lesion perhaps most frequently produced by these organisms is a gingivitis. When observed early the gums are somewhat swollen and injected and bleed readily on pressure. The commonest sites are adjacent to the rear molars. Overlying the swollen gums, there is usually a yellowish-white exudate which contains the characteristic spirochetes, fusiform bacilli and vibrios. This lesion, although frequently overlooked by the patient and by the physician, is a menace to the health, since from it the organisms may be aspirated into the lung and produce gangrene.

The important details of the cases of pulmonary abscess and of pulmonary gangrene observed at Mount Sinai Hospital in Cleveland in the past five years are presented in the following table.

ANALYSIS OF CHART

Multiple embolic pulmonary abscesses, manifestations of a staphylococcus septicemia or a pyemia, were observed in eight cases.

Abscess of the lung caused by pyogenic organisms but not associated with a pyemia or septicemia and apparently aspiratory in type was observed in nine cases, one third of which followed operation. Almost all were in infants or in children.

On the other hand, pulmonary gangrene occurred almost entirely in adults and three times as frequently as aspiratory abscess. About one half of the cases of gangrene occurred after operation.

In both aspiratory abscess and gangrene the lower lobes were more frequently involved than the others.

Six of nine patients with aspiratory abscess died, whereas eleven of fifteen with gangrene of the lung recovered. The striking results in gangrene, a much more severe process than abscess, were due to the

4. Smith, D. T.: Bronchopulmonary Spirochetosis, Am. Rev. Tuberc. **15**:352 (March) 1927.

5. Kline, B. S., and Berger, S. S.: The Relation of Oral Spirochetosis to Pulmonary Gangrene, J. Am. Dental A. **15**:64 (Jan.) 1928.

Cases of Pulmonary Abscess and Pulmonary Gangrene at the Mount Sinai Hospital, Cleveland
July, 1923, to May, 1928

Admissions to hospital	31,927
Major operations	5,803
Minor operations	14,436
General anesthesias	15,222
Local anesthesias	3,230
Total deaths	1,308

	Number of Cases	Sex		Age		Complicating Bronchitis or Pneumonia	Postoperative		Following Childbirth (Slight Ether Anesthesia)	Location					Arsenic Treatment		No Arsenic Treatment or Treatment When Moribund (Within Four Days of Death)		Postmortem Examination
		M	F	Infants and Children	Adults		General Anesthesia	Local Anesthesia		RU	RM	RL	LU	LL	Recovered	Died	Recovered	Died	
Staphylococcus septicemia or pyemia with embolic pulmonary abscesses	8	..	..	4	4	..	.....	....	.....	..	..	..	..	..	..	..	1	7	2
Pulmonary abscess (aspiratory)	9	7	2	7	2	6	1 oral operation; 2; 1 nonoral operation	1 nonoral operation	.....	1	1	2	1	3	..	..	3	6	3; 1 associated with B. pyocyaneus septicemia
Pulmonary gangrene (aspiratory)	25	24	9	3	30	11	5 oral operations; 12; 7 nonoral operations	1 nonoral operation	1	5	3	10	1	12	11	4	..	8	5
Pulmonary spirochetosis (aspiratory) before stage of ulceration	8					7	1 nonoral operation								7	..	1		

early recognition of the disease as gangrene and to the prompt and intensive treatment with arsphenamine and arsphenamine preparations.

CONCLUSIONS

1. Pulmonary abscess and pulmonary gangrene are separate entities. In spite of distinguishing characteristics, they are, however, almost universally considered and treated as one disease. At the Mount Sinai Hospital in Cleveland in the past five years, pulmonary gangrene has been observed three times as frequently as pulmonary abscess.

2. Pulmonary gangrene, although comparatively a much more severe process than aspiratory abscess, if proper treatment is used, offers an even better prognosis than does abscess.

3. There is conclusive anatomic and experimental evidence that pulmonary gangrene is caused by a group of organisms, notably spirochetes, fusiform bacilli and vibrios, aspirated from the oral cavity.

4. In cases observed clinically, careful examination of the sputum is sufficient to make possible a differential diagnosis between pulmonary abscess and pulmonary gangrene. In cases of pulmonary abscess, the sputum is whitish yellow, mucopurulent or purulent, without appreciable odor, and when washed shows pyogenic organisms, usually staphylococci. In cases of pulmonary gangrene, on the other hand, the sputum is foul smelling, grayish brown or grayish green, and when carefully washed and properly stained shows the characteristic spirochetes, fusiform bacilli and vibrios.

5. As the patients with pulmonary gangrene do not respond well to the treatment for abscess but are frequently cured by arsphenamine therapy, the differentiation between these two diseases is imperative.

6. Spirochetal pulmonary gangrene may be prevented by proper oral hygienic or therapeutic measures.

ABSTRACT OF DISCUSSION

DR. F. B. BERRY: At Bellevue Hospital, in New York, in Dr. Miller's service, we have had about 126 cases of pulmonary abscess; we have limited that term so as to apply only to abscesses or cases in which the abscess was purely in the parenchymatous tissue, nonbronchiectatic cases. More than 55 per cent of the patients have been cured by postural drainage alone. The results of treatment with arsphenamine have not been so satisfactory. In order to make a just comparison, it seem to me that only those cases should be considered in which arsphenamine alone was used without the employment of drainage, external or by posture.

Our results of needling have been somewhat similar to those reported by Dr. Young. In fact, wherever the diagnosis is made, we never needle, because we feel that not only the danger which Dr. Young expressed is present, but also other dangers too well known, and we consider it a most dangerous procedure.

DR. MILLER: Regarding the first part of the presentation in which the differentiation between the mythology of gangrene and abscess was presented, our

conception of this is that in almost all cases of suppuration of the lung which are included in this group, at one time or another we obtained evidence of these anaerobic infections, not necessarily caused by spirochetes, but usually by them and other organisms, just as has been described. With the suppuration, naturally because of the nature of the anaerobic infections, certain amount of necrosis developed. We have treated with arsphenamine between forty and fifty of the 126 patients of whom Dr. Berry spoke, all of whom raised foul-smelling sputum which contained spirochetes and other organisms, in many cases early; the results were disappointing. The number of cases in which the response was favorable was negligible.

DR. PRITCHARD: Regarding specific treatment for abscess of the lung, I think it makes some difference in spirochetal infections whether treatment is given early or late. In Dr. Miller's series many of these cases were what we might call chronically chronic. On the other hand, most of the cases that Dr. Young showed here were in the early stages of the infection—in some it had been present only a few days or a few weeks—and I think that is a point which should be considered in the good results and in the unsatisfactory and disappointing results in the treatment of spirochetal infections.

DR. GRAHAM: I would like to ask Dr. Young a question or two. Dr. Singer and I have had the experience, a number of times, of seeing the spirochetes and such organisms disappear from the sputum after the use of arsphenamine, but the patient, nevertheless, has not recovered from the disease which he apparently has. We have seen this happen repeatedly in the same person; that is to say, after treatment with arsphenamine the spirochetes disappear, but after some time they reappear, this is again followed by disappearance. I was particularly interested in the point which Dr. Pritchard has just mentioned in regard to these infections, namely, about the results of arsphenamine therapy in the experience of Dr. Kline and Dr. Young in chronic cases associated with spirochetal organisms.

DR. ELOESSER: In a recent case of actinomycosis at the San Francisco Hospital, the sputum swarmed with spirochetes. I would like to ask Dr. Young how much in some instances, she considers this spirochetal content of the sputum to be a fortuitous contamination.

DR. COLE: I feel a hesitancy in discussing abscesses of the lung. For the last three or four years, we have been interested in this condition and have had about forty cases. I have been making a study of the sputum and have been going to New York and watching Dr. Miller use neoarsphenamine. I decided to try it as a routine measure, but did not get any results. I tried postural drainage and pneumothorax and some other methods of treatment, each of which looked promising. I recently tried vaccine in large doses from 2 to 3 cc. twice a day; some abscesses of the lung cleared up that had not cleared up otherwise. I am interested to know whether the vaccine has been used or is being used, and if there is any other factor than the arsphenamine in this group of cases.

DR. YOUNG: In closing the discussion, I might start by apologizing for being here in place of Dr. Kline. He has been so enthusiastic and energetic in this work that I am sure he would have been able to tell you much more than I can. However, many of the discussions here have been similar to remarks and discussions that have been made at other times when Dr. Kline has been present, and I have had the opportunity on those occasions of hearing him answer.

In reply to Dr. Berry, I would say that Dr. Kline makes a distinction between abscess and gangrene on a bacteriologic basis rather than on the basis of the distribution of the lesion. That is, if there is an area of suppuration anywhere in

the lung produced by staphylococci with a sputum which is yellowish and not foul smelling, he considers the lesion an abscess; whereas, if it is an ulcerated area with green, foul-smelling exudate and shows the characteristic spirochetes, fusiform bacilli and vibrios, he calls the lesion gangrene.

Dr. Pritchard answered Dr. Miller in saying that successful cases are the ones in which treatment is given early. In their paper, Dr. Berger mentioned that he believed the good results he obtained at Mount Sinai Hospital were due to early diagnosis. Another factor of importance is the amount of neoarsphenamine given. When the patient is extremely ill and coughs up large quantities of green, foul-smelling sputum, intensive treatment is necessary. Intravenous injection of 0.9 Gm. of neoarsphenamine every seventy-two hours is more effective in these advanced cases. However, the best results are obtained in the early cases.

In regard to Dr. Graham's question about the spirochetes disappearing from the sputum, I would like to say that we have had a great deal of difficulty in getting satisfactory specimens of the sputum in the laboratory. When a patient knows that you want a specimen he usually tries to oblige, and coughs and produces a secretion from the throat rather than exudate from the involved lung. Variable results follow if some of the specimens are unsatisfactory. These organisms are very fragile, and on standing at room temperature over night or for twenty-four hours, examination of the sputum may not reveal the characteristic organisms.

Several discussers have mentioned the possibility that the spirochetes and fusiform bacilli are saprophytes superimposed on some other organism. I showed one case in which gangrene was superimposed on tuberculosis. Dr. Kline's reply to this question on one occasion was, "If these organisms are saprophytes, then God deliver me from saprophytes." The final proof of whether spirochetal pulmonary gangrene is due to spirochetes, fusiform bacilli and vibrios will be obtained when the organisms are isolated in pure culture and the characteristic lesion is produced in animals inoculated with pure cultures. At the present time Dr. Kline is having built in the laboratory a jar in which he can produce anaerobic conditions and obtain pure cultures of these organisms.

In answer to the last question as to whether they are the actual causative organisms, Dr. Kline believes that they are and expects to get positive results with pure cultures of these organisms. One basis for this belief is the observation that at the periphery of progressively enlarging gangrenous cavities spirochetes are present in enormous numbers, and in places are the sole organisms present.

SURGICAL PRINCIPLES UNDERLYING ONE-STAGE LOBECTOMY

HAROLD BRUNN, M.D.
SAN FRANCISCO

It seems remarkable that so few articles or discussions are to be found during the last few years on lobectomy. The discussion has rather concerned the substitutes that might be used in its stead, based no doubt on the theory that the operative mortality forbids its use or that the technic of the operation has become a fixed procedure.

Lobectomy, that is, the removal of one or more diseased lobes of the lung, must appeal to every one as the ultimate goal in surgical procedures on the chest. By this method the diseased lobe is removed at one stroke; the period of convalescence is diminished, and deformity does not result. The method also more nearly conforms to the procedures surgeons are accustomed to apply for diseases in other organs of the body. Just as cholecystectomy superseded cholecystotomy and as hysterectomy superseded the old time method of extraperitoneal treatment of the uterine stump for fibroids, may one not also expect in the future that with improved technic and lowered mortality lobectomy will become the operation of choice in many diseased conditions of the lung?

My own experience with the operation, meager though it is, has brought out several points of value which I think are worthy of discussion. I give my own technic for what it is worth with the hope that it may bring out further opinions and finally greater improvement in this operative procedure.

I am reporting in this paper six cases in which lobectomy was performed with one death; the operation was for bronchiectasis in five of the cases and for malignancy in one, with removal of the lower and middle lobes. Perhaps luck was with me. I am sure that has been so in some of the early cases as I look back on them, because there has been a gradual change in the technic which I believe is a considerable improvement over the previous methods. My own interest in lobectomy was stimulated by the writing of Dr. Howard Lilienthal whose method has been the basis on which I have done all my work, modified from time to time as experience seemed to direct.

Since bronchiectasis is probably the disease for which lobectomy is most commonly advised, a word may be said as to the operative indications. As the histories will show, the condition of the patients was serious, and there was a prospect of chronic invalidism for life. It was evident that there was no prospect of otherwise ameliorating the condition to any extent or with any degree of permanency by the usual

methods of treatment. I have, however, in my work made one definite distinction. In the last few years it has been common for surgeons to use the term "chronic lung suppurations," which is made to include not only true bronchiectasis, but other forms of suppurations from the lungs, especially chronic multiple abscesses. I believe that from either standpoint this has no real advantage. From my own experience, I feel that these multiple chronic suppurative processes have for the most part a different beginning. The parenchyma rather than the bronchi is the seat of the disease, which extends or breaks into the bronchus rather than from it. I believe that these processes run a somewhat different course with more frequent acute exacerbations, that with iodized oil 40 per cent the bronchi do not fill as readily or in the same way as in true bronchiectasis, and that in the surrounding lung there are frequently abscesses, not shown by x-ray examinations, that have not as yet broken into a bronchus. It is my opinion that except in carefully chosen cases, there will be a higher mortality when lobectomy is performed than there will be in the usual cases of bronchiectasis. I believe that both pathologically and clinically and from the standpoint of treatment they are different entities, and that one should endeavor to discriminate between these different pathologic processes as far as possible rather than to group them under one general heading, since they have so many distinguishing features.

OPERATIVE PROCEDURE

Preliminary Treatment.—I choose for operation patients whose lungs are seriously involved without prospect of cure but who are not hopelessly septic. The preliminary treatment extends over several months and usually consists of rest in bed, exposure to sunlight, postural drainage, the use of the bronchoscope, cleaning up of the sinuses and possibly the use of vaccines. In cases of spirochetal infections, neoarsphenamine is used. I attempt to have little or no acute parenchymatous involvement of the surrounding lung. With the preliminary treatment, the patients can attain a fairly good state of health with an increased vital capacity and increased resistance. I usually give 500 cc. of dextrose solution intravenously the night before operation. I seldom give a blood transfusion. From 1,500 to 2,000 cc. of Ringer's solution is injected subcutaneously into the muscles of the thighs continuously while the operation is being performed.

Phrenicotomy.—While I have not used phrenicotomy as a preliminary procedure in the cases reported, I believe that it has advantages. The phrenic nerve may be severed in the neck two or three weeks before the major operation, or, if one chooses, it is easy to sever it on the left side as it courses over the pericardium. The cutting of the phrenic nerve

will keep the diaphragm quiet during the operation and following it will diminish the size of the cavity left after the removal of the lobe by its increased elevation. I shall adopt this procedure in the future.

Anesthesia.—The patient is placed on the table with the head low in order to favor drainage and do away with tracheal suction, although this may be used if necessary. In my early cases I used intratracheal anesthesia, then intrapharyngeal, and later positive pressure with gas and oxygen (Bunnell apparatus). I believe that the present method is a considerable improvement, and that the patients are subject to much less shock.

The morning before operation, postural drainage is instituted. A barbital suppository of 15 grains (0.972 Gm.) is used, or 10 grains (0.65 Gm.) of barbital is given by mouth from three to four hours before operation and $\frac{1}{200}$ grain of scopolamine (0.3 mg.) and $\frac{1}{4}$ or $\frac{1}{6}$ grains of morphine (16 or 11 mg.), depending on the body weight and strength, is given one and one-half hours before operation. One half of this dose, $\frac{1}{400}$ grain of scopolamine (0.15 mg.) and $\frac{1}{8}$ grain of morphine (8 mg.), is repeated half an hour before operation. A syringe is loaded for the operating room containing $\frac{1}{400}$ grain of scopolamine and $\frac{1}{8}$ grain of morphine to be used at any time during the procedure. Procaine hydrochloride, 0.5 per cent, with a drop of epinephrine to the ounce is injected in rather large quantities along the intercostal nerves, both above and below the line of incision. I think that the suggestion of Eloesser that 3 or 4 cc. of 4 per cent procaine hydrochloride with 5 drops of epinephrine be dropped down the trachea before operation, as is done before the injection of iodized oil, may have advantages in preventing cough. I believe, however, that scopolamine and morphine are the most valuable drugs to allay coughing, and since I have used it I have had little difficulty in keeping the patient quiet. The patients are more or less obtunded, and even the most nervous person sleeps at least half way through the operation, awakening to complain only when some more painful part is dragged on. It is important to adjure the patient not to cough during the operation. This should be done for several days before and be impressed on him all the time. In my experience, it is effective. The barbital is used as an added hypnotic for its synergistic effect and because of its antidotal properties to cocaine and all its derivatives. For those patients who have a low blood pressure or whose blood pressure begins to fall 1 cc. of ephedrine is used. I believe this is the most useful drug that can be used in operations on the lung. The drug is sometimes given again at the end of the operation. I have used alpha lobeline as a respiratory stimulant, but do not feel that it is very effective. A tank of carbon dioxide and oxygen is kept at hand to be used as a respiratory stimulant. This, no doubt, is the most effective measure for the purpose.

When these measures have been taken, I feel fairly well armed against the ordinary untoward events that may occur during the operative procedure.

With the patient under this type of anesthesia, after the preliminary intercostal incision has been made, a small opening is made in the pleura, and with the finger over it, the air is allowed to enter slowly. At the same time the patient is adjured not to cough. The lung will collapse gradually. I have never found it necessary to fill the chest with air for a week before operation, as is recommended by Eloesser. By this method, I have opened many chests for various conditions, and although I have carefully looked for changes in the pulse rate and respiration and for evidences of shock, I have never noted any disturbance; it is as if nothing unusual had occurred. Symptoms do arise, however, if the patient begins to struggle and cough. If this happens, I close the opening with a wet towel or drag on the lung if the chest is already opened, covering the opening as rapidly as possible, and allow the cough to pass off. At this time, the third dose of scopolamine may be given; I usually do not have any more trouble during the rest of the operation. The patients are free from pain. They sleep the greater part of the time, but can be aroused sufficiently to blow up the lungs by holding the nose if one wishes. The last case that I had was that of a young man, aged 21, nervous and fairly irascible, who nevertheless went through the operation without any difficulty.

Incision.—The incision I have used has followed the intercostal route between the seventh and eighth or sixth and seventh ribs, severing one, two or three ribs above posteriorly, as is recommended by Lilienthal (fig. 1). The only precaution is to enter the pleural cavity by making a small opening which is blocked by the finger in order to permit the air to enter slowly and to allow the lung to collapse gradually.

Mobilization of the Lung.—In my experience, adhesions have never been a serious difficulty in cases of bronchiectasis. For the most part, these adhesions have been between the diaphragm and the under surface of the lower lobe and at the fissures between the two lobes. The separation of adhesions from the diaphragm has usually been rapid, only now and again requiring clamping and tying. The separation of adhesions between the lobes frequently takes more time, requiring careful cutting between clamps, tying and sometimes whipping over the raw surfaces. It is wise to transfix the ligatures in this region, as otherwise they may slip when the upper lobes are expanded under pressure, permitting not only bleeding, but the escape of air into the pleural cavity. I make every effort to avoid the latter occurrence (fig. 2).

When adhesions are extremely dense, as I have found when a two-stage operation has been attempted, the procedure consumes much more

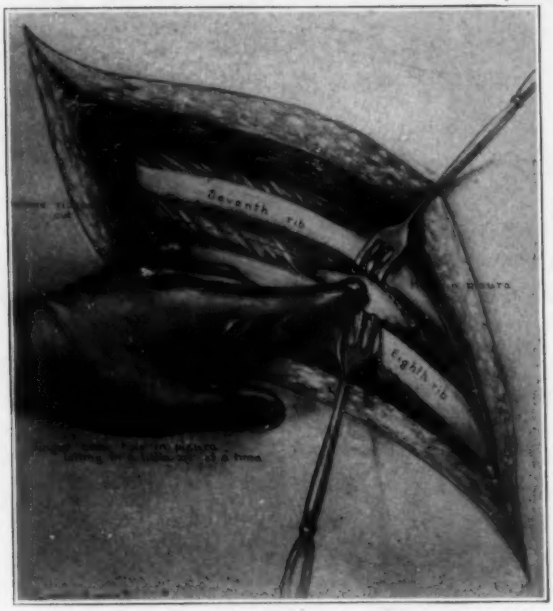

Fig. 1.—A long intercostal incision between the seventh and eighth ribs extending from the cartilages anteriorly to the rib angle. The dotted line shows where the ribs are severed posteriorly. A small nick has been made in the pleura over which the finger has been placed in order to permit the air to enter slowly and allow gradual collapse of the lung.

Fig. 2.—The ribs have been retracted with the Balfour retractor. The severed ribs posteriorly are shown and the working field developed. The upper lobes above are collapsed and the diseased lung somewhat thickened. Adhesions to the diaphragm and between the lobes have been separated to develop the pedicle.

time and is much more dangerous because of shock and the dislodgment of emboli which the increased handling entails.

The Pedicle.—After I have freed the adhesions on all sides and separated the lung at the fissure, my attention is next directed to the pedicle (fig. 3). The pulmonary ligament below is freed rapidly, and I attempt to make a pedicle as small as possible at the base, at the same time keeping away from the mediastinum. The pulmonary veins lie below the bronchus as a rule, and in freeing this part of the pedicle care is taken not to cut into them. In many cases, the bronchus can be felt, and it is

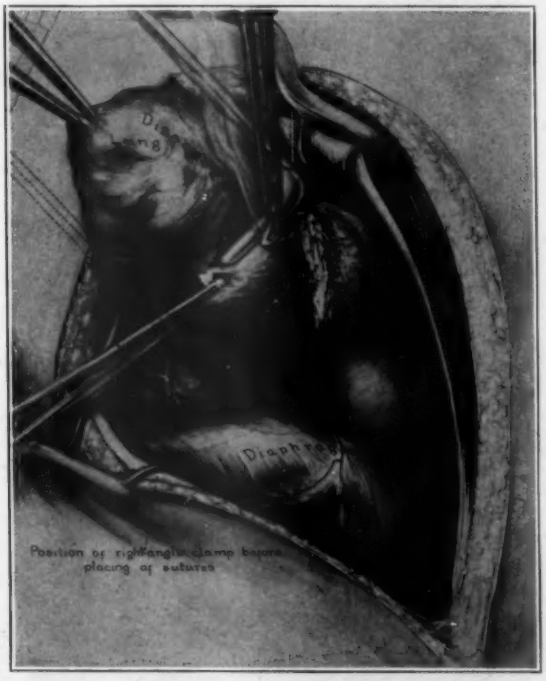

Fig. 3.—The pedicle has been clamped at the base and double interlocking chromic gut sutures tied. Cautery is cutting below the distal clamps leaving a liberal stump behind the ligatures.

not difficult to clamp the pedicle below the bronchus which carries the pulmonary veins. This step should be taken as soon as possible in the dissection; if the case is difficult, I do this before separating the lung at the fissure, as I believe that by clamping the vein first emboli are prevented from entering the circulation during future manipulations. The pedicle is then clamped on the upper side of the bronchus, and finally the bronchus itself. I use the right angle Wertheim hysterectomy clamps with rather long blades. A second set is placed distal to the first, leaving a sufficient pedicle of lung projecting between them. Double no. 2 inter-

locking chromic ligatures are placed in front of the proximal clamp above and below and around the bronchus. The clamps are removed and the pedicle tied in series. The ligatures are tied in the groove left by the removed clamps. With an actual cautery, a somewhat wedged-shaped incision is made in the lung, and at this time the projecting bronchus can be cauterized still further. A running stitch of chromic catgut is taken back and forth twice over this pedicle to prevent any further oozing, and especially to prevent any leakage of air through the bronchus (fig. 4). I consider the closure of the bronchus a most important step,

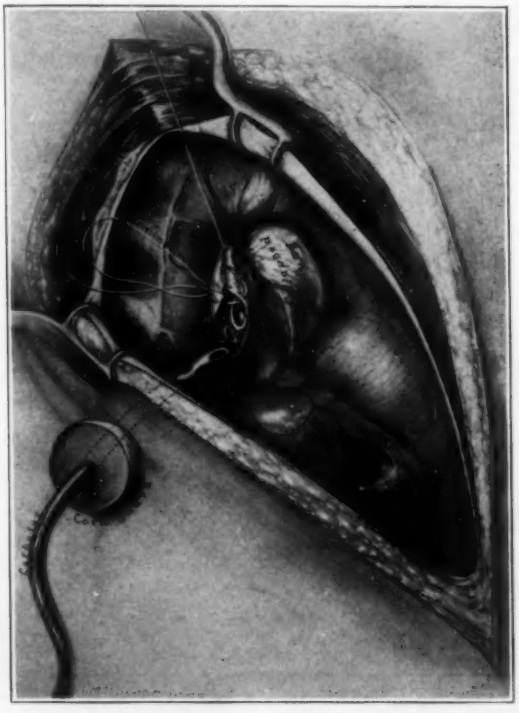

Fig. 4.—The pedicle is being closed over with a running stitch of chromic gut. The drainage tube has been inserted through the intercostal space below extending a short distance into the pleural cavity guarded by a cork to prevent leakage of air.

as it is my endeavor to keep the chest as free from leakage of air as possible during convalescence. It is sufficient, I think, to keep the cavity free from air for from five to seven days. I have not seen the necessity of using any special method in handling the stump. Some of the methods are time-consuming, and time is an element in these cases. The stump sloughs off or atrophies. I believe that leaving a liberal stump far enough away from the mediastinum causes less shock, and I have not seen any mediastinal infections. During all these intrapleural manipula-

tions, the wound in the chest has been covered with gauze pads and rubber sheeting as has the collapsed lung above. It has been noted how little infection of the pleura occurs following the operation. Working in the chest with the upper lobes collapsed makes the operative procedure rapid. Under other forms of anesthesia, the upper and middle lobes are in the way, and the anesthetist must constantly vary the pressure, consuming time and increasing the shock to the patient. At times the disease in the lung has produced such marked fibrosis that careful dissection cannot be made, and the pedicle must be clamped en masse.

Fig. 5.—Method of wound closure, the ribs being brought together. The muscles are sewed in layers and the wound is airtight.

Testing the Pedicle.—The pedicle is dropped back, the gauze is removed, the patient's head is raised somewhat at this stage, conditions warranting, and gas and oxygen anesthesia under pressure is now started. At the same time, the chest is filled with salt solution covering the pedicle and those parts of the lung above which have been severed. The test is made to determine whether the pedicle is airtight. If any bubbles of air are seen arising through the water, either from the lung above or through the pedicle, more stitching must be done to prevent leakage. Also, under added pressure and changed position, vessels may bleed, and

the blood can be seen in the salt solution. The chest may have to be filled once or twice in this procedure to make sure that the cavity is airtight. Now that the chest is dry and the pedicle airtight, the chest wound is rapidly closed in the usual way by chromic gut ligatures around the ribs, the wound being sewed up in layers; for purposes of drainage, however, a rubber tube is first placed below the line of incision through a cannula (fig. 5).

Air and Fluid in the Pleural Cavity.—I believe that the progress of the patient depends on how thoroughly the chest cavity is kept free from air and fluid for the next five to seven days. If the pedicle is a success, if the wound does not leak air, and if by constant suction, either with the hand syringe every two hours day and night or by some form of suction apparatus, the chest cavity is kept free from air and fluid and the upper and middle lobes are allowed to expand, the course of convalescence is usually mild and almost free from shock.

Bronchial Fistula.—There is no permanent bronchial fistula. I believe, therefore, that a special method need not be used to take care of the pedicle. To prevent hemorrhage and the leakage of air through the bronchus from five to seven days, one of several methods may be employed, the simplest being the best. After this, the pedicle sloughs away and the bronchus opens, although it may be delayed for two or three weeks. By this time the upper lobes are expanded and adherent, and the temporary fistula is of no moment. Bronchial fistulas, in my experience, are exactly like fecal fistulas. They remain patent only when they are superficial and attached to an unyielding wall. Deep fecal fistulas, as every surgeon knows, always close when let alone; the same may be said of bronchial fistulas. I am even more averse to the use of gauze packing in surgical procedures on the lung than I am to the use of such packing in operations on the abdominal cavity. I believe that it has but one use, and that is for control of hemorrhage in an emergency. I hold in particular disfavor the placing of gauze against a pedicle. It tends to loosen the ligatures and create pockets of infection. The pleural cavity seems to be able to take care of infections as efficiently as the abdominal cavity, if at the termination of the operation it is left in as physiologic condition as possible.

Convalescence.—The patient usually leaves the table in good condition, but secondary shock often occurs three or four hours later. For this the patient receives ephedrine, salt solution and dextrose solution. Suction is employed to keep the chest free from fluid, and efforts are also made to keep it free from air. A great quantity of fluid collects in the pleura at this time; this fluid should be only slightly blood-tinged. Dark red blood means hemorrhage and necessitates blood transfusion and reopening of the wound. Otherwise, the state of shock rapidly passes

off, and in the morning the patient usually looks as well as after an ordinary operation. Suction is continued day and night every two hours from the tube in the chest. In two or three days, an x-ray picture is taken to see whether or not the upper and middle lobes have expanded. If the operation has been successful, the lobes will be found expanded and already attached to the visceral pleura by some adhesions, but I usually feel that from five to seven days are required for them to become fixed in that position. During these days, the fluid does not become purulent, and it has no particular odor. One is surprised that the patient's temperature is practically normal. I have never seen any vicious infection of the pleural cavity, and in my later cases the wounds in the wall of the chest have all healed by first intention. I have left merely a small pocket as large as an orange where the posterior lobe was removed with the upper and middle lobe now fixed by adhesions to the wall of the chest above it. At this time—from five to seven days after the operation—I begin the use of a surgical solution of chlorinated soda (Dakin's solution). Gradually, in the course of two weeks, the discharge becomes more purulent due to the sloughing pedicle. If there is now a secondary rise of temperature, I remove one or two ribs for drainage.

I appreciate that the number of patients subjected to this operation is altogether too small to base conclusions on their cases. Nevertheless, the rapid and satisfactory convalescence of some patients leads one to believe that this operation should receive wider consideration in lung surgery. No doubt the other procedures now in use will have their place in the individual case, but now and again conditions ideally suited for lobectomy will be found at the operation table in every chest clinic, and I believe that this measure should not be discarded as it seems to have been in the past.

The factors which make for success in one-stage lobectomy are: careful preparation of the patient, local anesthesia with scopolamine and morphine preceded by barbital, careful hemostasis and, most important, the prevention of pneumothorax and hydrothorax for at least five days following the operation. I feel that if I succeed in carrying out these measures satisfactorily in my cases I may look forward to an easy immediate recovery of the patient and a short convalescence, neither fistula nor deformity occurring.

REPORT OF CASES

CASE 1.—My first operation was performed in January, 1918, on a man, aged 26. With the patient under intratracheal anesthesia, the technic of Lilienthal was employed in removing the lower lobe of the right lung. There was also involvement of the same lobe in the left lung. Convalescence was prolonged and stormy, and several secondary operations were necessary. He left the hospital improved, but was lost track of and never returned to report.

CASE 2.—*History.*—C. W., referred by Dr. T. C. Edwards of Salinas, was admitted to the hospital on Aug. 2, 1918, and discharged on October 6, a resection of the lower lobe of the right lung being performed. The patient complained of fetid breath and the expectoration of a great quantity of a foul smelling sputum, about three or four cupfuls a day. The father, aged 55, was living and well; the mother, aged 48, was also living and well. He had three sisters, aged 17, 23 and 25. One sister died in infancy from scarlet fever. One brother was living and well at the age of 21. There were no tuberculosis, mental or malignant diseases in the family. The past history showed that the patient was born in San Francisco and had never been outside of the state except to Arizona. He seldom used coffee or tea, and never used alcohol or tobacco; the only drug used was creosote for cough. He had been a strong, healthy baby. He had measles at 3 years of age, scarlet fever at 5, chickenpox at 6, whooping cough, one attack, at 5½ and typhoid (?) at 15 years of age. He did not have frequent colds during the winter, and felt well, strong and healthy until 1915. He said that he had not had gonorrhea or syphilis.

The present illness began about the middle of October, 1915. While apparently in good health, the patient acquired a sore throat and felt weak. Fever developed. with pains in the joints and back. After three days a physician was consulted, who diagnosed the condition as diphtheria. Antitoxin was given and resulted in some improvement. Four days later, he became worse; he had high fever and began to cough up quantities of yellowish-green sputum with a very bad odor. This was occasionally blood-tinged, but there was no frank hemorrhage. After a month, by November, 1915, the general condition had improved so that he was able to walk about, but the cough continued. At this time, he had lost 20 pounds (9 Kg.). In December, his condition became worse, and it was at this time that I saw him. An x-ray picture by Dr. Painter showed large bronchiectatic cavities in the lower lobe of the right lung. The patient was septic and cadaverous, having a high fever. He was advised to return home, to use postural drainage and to live outdoors. I felt, however, that his condition looked hopeless in every way. He returned to me again in 1918, and entered the Mount Zion Hospital. At this time he was a large, strapping man, 22 years of age, and weighed 175 pounds (79.4 Kg.). He was tanned from living outdoors, and the disease had evidently become chronic. There was no fever, but he still persisted in coughing up large quantities of sputum, from 200 to 300 cc. a day, which was very foul-smelling. On account of the odor of the sputum, he was unable to obtain work. No one cared to associate with him, and he begged us to do something to relieve him of his condition. X-ray pictures taken at this time showed the major part of the process in the lower lobe of the right lung, but there was a suggestion of cavitation also on the left side (fig. 6*A*). On his earnest solicitation, it was decided to operate on the lower lobe of the right lung, in the hope of giving him at least partial relief.

Operation.—Operation was performed on Oct. 8, 1918, at Mount Zion Hospital. Intratracheal anesthesia was given by Dr. Botsford. A long intercostal incision was made from front to back between the seventh and eighth ribs, as described by Lilienthal, which gave plenty of exposure. There were few adhesions. The diseased lower lobe was noted lying in the lower part of the chest, and was distinctly different from the middle and upper lobes. It was red, mottled and firm to the touch. There were a few adhesions to the diaphragm, but these were easily freed. The pedicle of the lung was mobilized below, and the lower lobe was separated from the middle and upper lobes. The root of the lung was clamped with right angle Wertheim clamps. The pedicle was tied off with double no. 2 chromic catgut in series. Dissection of the lung was made beyond the ligatures with the

cautery, and the end of the stump was closed with a chromic gut lock stitch. A small tube was left between the ribs below the incision. The chest was closed tightly in layers. The tube which was passed into the chest was attached to the suction apparatus as soon as the patient was put into bed and negative pressure was kept up constantly.

The operation progressed smoothly, but after its completion the patient was in a state of shock, and it was necessary to stimulate him and give fluids under the skin. Thereafter he had a stormy convalescence and empyema developed as was expected. Surgical solution of chlorinated soda (Dakin's solution) was used. It took him four months to recover. For a time bronchial fistula persisted, but a secondary operation was not necessary for its closure. The wound closed without any deformity of the chest, and while the patient was never free from cough, he went home in a much improved condition (fig. 6 *B*).

The bacteriologic report concerning the pus from his lung showed *B. aerogenes* to be the predominating organism. There were two types of nonhemolytic strep-

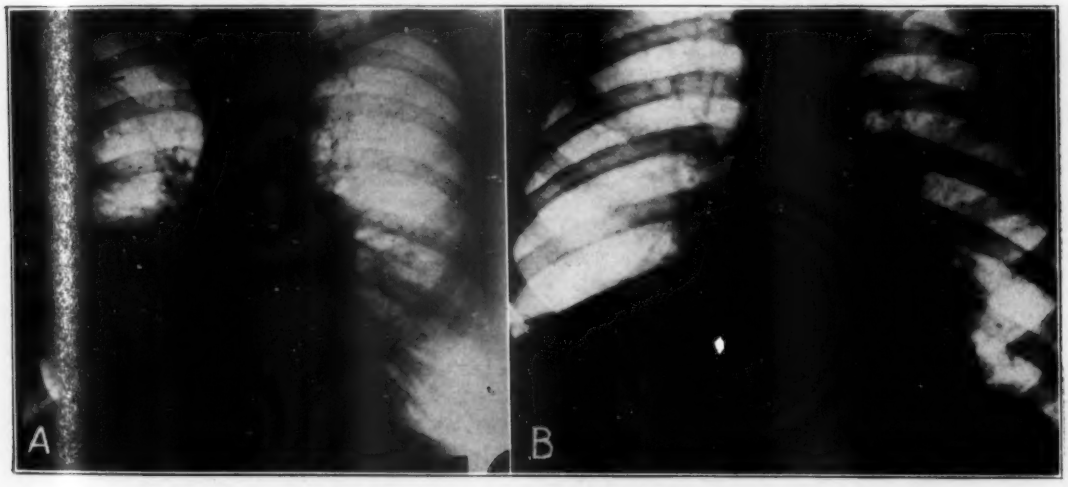

Fig. 6 (case 2).—*A* shows bronchiectasis of both lobes, more marked on the right, in C. W., before operation in 1918; *B*, one year after removal of the lower lobe showing elevation of the diaphragm. The lung is expanded.

tococcic organisms (one green pigment-producing on blood agar, the other not). A period of improvement followed, but without entire disappearance of cough and expectoration. In time the patient married, went to work, and seemed to be getting along very well. An x-ray picture taken in September, 1921, showed enlarged bronchial root glands with diffuse peribronchial thickening; multiple rounded masses of increased density at the base of the left lung, and a triangular shadow of increased density at the base of the right lung. As time went on, there was more and more expectoration. The disease on the left side progressed rapidly. The patient again began to cough up large quantities of sputum, and he finally committed suicide four years after the operation, on Aug. 23, 1922.

Microscopic Examination.—A specimen examined on Aug. 9, 1918, consisted of a large portion of the lower lobe of the lung, naturally somewhat torn and compressed in operative removal. On section, the conspicuous observation was the presence of a number of greatly thickened, whitish, tubelike structures, evidently

greatly thickened bronchi with hyperemic mucous membrane coursing through the contracted tissue. There also occurred cystlike spaces which attained the size of a robin's egg. These showed the same structure as the bronchi, except that the wall was thinner in the dilated portions. Vessels with walls thickened by surrounding connective tissue were also seen. The intervening parenchyma of the lung was mostly atelectatic, soft and hyperemic, but did not give the fulness of an organized pneumonia. Sections from various portions showed greatly thickened bronchi with convoluted epithelium, the individual cells being enlarged considerably in places; beneath this layer was a dense colony of lymphoid and epithelial cells, and surrounding this a varying amount of connective tissue. These structures so splinted the adjacent lung tissue that it had prevented contraction and expansion to an extent that the epithelium in the adjacent alveoli had reverted to the cuboidal form. The vessels adjacent to the bronchi showed varying grades of chronic endarteritis. The smaller bronchi showed a similar reaction, and the fibrous tissue irregularly infiltrated the interalveolar septums.

The alveoli showed edema in places and frequently contained vacuolated, desquamated epithelial cells. The chronic fibrous reaction was limited to the interalveolar structures, and the tufts of ingrowing connective tissue of organizing pneumonia were not observed. In no region was anything observed to indicate a specific infection as the basis of the process.

A diagnosis of chronic proliferative bronchitis with marked bronchiectasis was made (G. Y. Rusk).

Case 3.—*History.*—I. M., aged 26, married, an American housewife, was referred by Dr. Anson Hill. She had had bronchitis frequently from infancy to the age of 8. In 1922, she had severe influenza and pneumonia. In the fall of 1918, she had an operation for suspension of the uterus and an appendectomy. Pneumonia followed the operation. The patient dated her trouble from that time, and said that she had had a persistent cough since then.

Eight months after the operation, she developed a severe pain in the right side of the chest at the level of the fifth rib. After a few days, she began to cough up quantities of foul-smelling sputum mixed with blood. The pain simultaneously disappeared. She had a morning cough lasting for about an hour and raised from 400 to 600 cc. of sputum. She had slight cervical adenopathy; marked clubbing of fingers, and amphoric breathing on the right side.

The urinalysis and Wassermann reaction of the blood were negative. The sputum was negative to acid-fast organisms and elastic fibers. Culture showed *B. mucosuscapsulatus.* X-ray examination showed increased bronchial tree markings of the upper lobe of the right lung with pleural interlobular thickening. There was mottling at the base of the right lung with peribronchial thickening suggesting bronchiectasis (fig. 7 *A*). In the hospital, the temperature varied from 98 to 99.5 F.; the pulse rate, from 80 to 110, and respiration, from 20 to 25.

Operation.—Lobectomy was performed on April 6, 1923, on the lower lobe of the right lung. Procaine hydrochloride and gas oxygen anesthesia was used. A long intercostal incision was made between the seventh and eighth ribs. The lung was free; adhesions were not found either above or below. The diseased lobe expanded under positive pressure; it looked pink and normal, and for a moment there was doubt as to whether or not there had been a mistake as to the side that required operation. Convinced that the lobe was not the one that was diseased, I mistook the diaphragm for a possible lobe and attempted to make a dissection of it. I soon found my error, however, and desisted, but not before I had separated the diaphragm partly from the side of the wall of the chest. I then returned to

the lower lobe, and, grasping it with a lung forceps, I was able by a little dissection to make a small pedicle. This was clamped with a Wertheim right angle clamp. The pedicle was tied off with interrupted double chromic no. 2 catgut. The lung was burned off and a running stitch applied as before described. In this particular case, I was extremely careful to close the bronchus. I protected the edges of the wound and the surrounding pleura with gauzes and rubber dam during the cutting of the pedicle. There was no bleeding. The patient did not give any trouble on the table, but the positive pressure on the upper and middle lobe at times interfered with my manipulations. I had an interesting experience as the result of the injury to the diaphragm. The morning following the operation, the intern drew my attention to a tremendous swelling on the right side of the abdomen and flank. I realized immediately that air had apparently seeped down from the chest extraperitoneally. By putting pressure on this and increasing the suction, it rapidly disappeared. Fortunately, infection did not occur in this region, so that it gave no trouble, although for a week I was anxious about this possibility. Suction was kept up persistently every two hours for four or five days. At that time I considered that the upper lobes had securely fastened themselves to the wall of the chest and irrigations were now begun with a surgical solution of chlorinated soda to combat the beginning empyema. This patient, except for a short time during the afternoon when she was somewhat shocked, made an excellent convalescence. The following morning she did not show any more effects from her operation than from an operation for ordinary hernia, and she continued to make a rapid recovery (fig. 7*B*).

On June 1, the patient was well, except for a small cavity the size of a lemon where a pneumothorax still persisted and a bronchial fistula. On this date, I removed several ribs, closed the bronchial fistula, separated some of the adhesions of the upper lobe to allow it to expand and covered the fistula. The patient made a good recovery, and was discharged from the hospital a month later with the wound entirely healed. At the time of operation, I recognized the possibility of the presence of bronchiectatic cavities in the middle lobe, but because of making a mistake concerning the lower lobe, which was normal in appearance, I did not feel justified in removing both lobes at this time. This operation was performed before the use of iodized oil.

Two years ago, I injected the patient's bronchial tree with iodized oil solution and found some small pockets in the middle lobe and some fine dilatations in the upper lobe of the same side (fig. 7*C*).

Recently another injection of iodized oil was made. On this occasion I did not see the dilatations in the upper and middle lobes of the affected side, but there were distinct enlargements in the lobes behind the heart. The patient has, however, remained well. She is plump, has a good color and does her own housework. She has had few colds and no sinus infection. She raises about a tablespoonful of sputum in the morning which is not purulent. She feels well satisfied with the results of the operation.

Examination of Specimen.—The gross specimen examined on April 7, 1923, consisted of the lower lobe of the right lung; the lobe appeared somewhat small, and the tissue felt collapsed; the external surface, particularly the lateral aspect, was roughened by fibrous adhesions; mesially, the main bronchial branch was seen partly opened and showing thickened walls lined by a grayish wrinkled mucosa; adjacent to this, and below near the inferior surface of the lobe were from three to four large, elongated saclike structures that ended blindly; they had thickened fibrous walls and a wrinkled granular lining similar to that of the bronchus. Sec-

tion through the entire lobe showed one or two other dilated thickened bronchial branches near those described; otherwise, the tissue presented a normal pinkish-gray appearance without consolidation, fibrosis, cavitation or other pathologic processes.

The microscopic examination of sections through the region of the main bronchial branch showed this to be lined by a markedly thickened mucosa, thrown

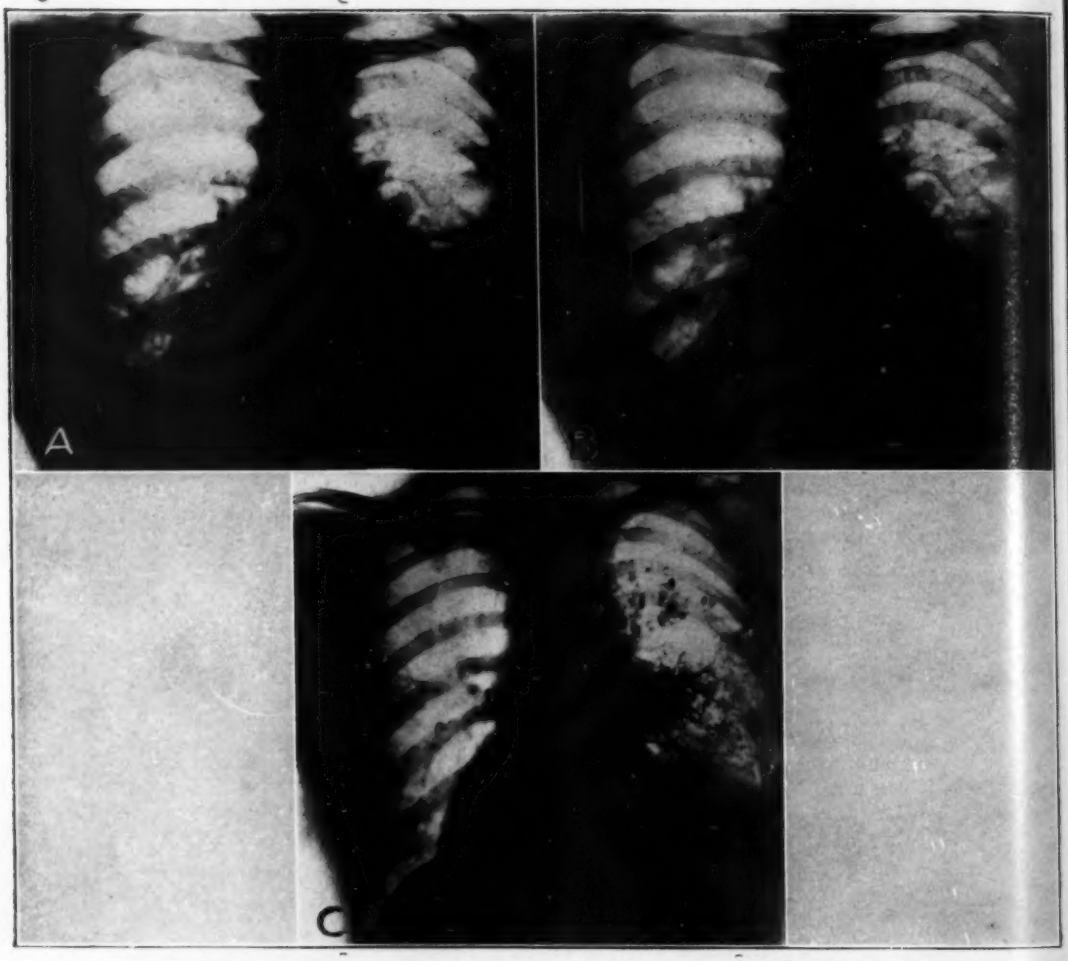

Fig. 7 (case 3).—*A* shows bronchiectasis of the lower lobe of the right lung in I. M. in April, 1923; *B*, about three months after operation, and *C*, after an injection of iodized oil, Sept. 23, 1924, showing cavitation at the hilum of the middle lobe and a few small dilatations in the upper lobe.

into numerous folds, the latter covered by a fairly well preserved pseudostratified layer of ciliated columnar cells, the subepithelial connective tissue infiltrated by tremendous numbers of plasma cells and lymphocytes, the cellular infiltration extending to a moderate degree into the underlying mucous glands. Cross-sections of smaller bronchioles were seen about which a similar infiltration was present.

Except for a slight infiltration of similar cells about some of the blood vessels, the limitation of the infiltrate strictly to the connective tissue immediately surrounding and forming the walls of the bronchioles was striking. The bronchiole lumina contained a small quantity of pus and cellular debris. The adjacent alveolar spaces appeared exceedingly thin-walled, somewhat collapsed, and practically entirely empty. The intervening alveoli were small, empty, lined by low cuboidal cells, large cavities incompletely lined by a slightly rugous, stratified, squamous epithelium, supported by a thick layer of vascular granulation tissue containing large numbers of plasma cells and lymphocytes and some eosinophils. At one place the epithelial lining was of the ciliated columnar cell type. The sac lumina were empty. The intervening alveoli were small, empty, lined by low cuboidal cells, their walls thickened with fibrous tissue and with a moderate infiltration of lymphocytes and plasma cells. Some of the blood vessels showed marked thickening of the intima. Evidence of tuberculosis was not found. The gross specimen was preserved.

A diagnosis of bronchiectasis of the lower lobe of the right lung was made.

CASE 4.—*History.*—J. M., aged 26, was first admitted to the hospital on May 11, 1923, and discharged on November 28. He complained of cough with hemoptysis and hematuria and pain in the region of the right kidney. The family history was negative. The past history showed that the patient had pneumonia in 1916 for three months and influenza in 1918 for two months. He had no other serious illnesses. The present illness began when the patient had influenza in 1918, which was followed by a chronic productive cough with occasional severe pains in the left side of the chest suggesting pleurisy. He had several rather severe hemorrhages shortly after he began to have a chronic cough. He had lost 17 pounds (7.7 Kg.) in weight. He worked most of the time in spite of his illness. Six months before admission to the hospital, he began to have dull pain in the region of the right kidney, accompanied by hematuria. The hematuria lasted only two days. Two weeks before, he again developed hematuria and simultaneously had considerable hemoptysis. He was admitted to the tuberculosis service and was thought to have pulmonary and renal tuberculosis.

Physical examination showed râles and amphoric breathing at the base of the left lung. The right lung was clear. The heart was normal. The abdomen was slightly tender in the region of the right kidney. There was a large right inguinal hernia descending into the scrotum. According to the x-ray report, the process in the left lung might be old pneumonia or metastatic carcinoma.

Operation.—On July 13, 1923, the right kidney was removed. There was an irregular tumor occupying the lower half. Microscopic examination showed adenocarcinoma apparently arising from tubules in the cortex. Evidence of extension was not found at operation. Convalescence was uneventful. The chronic cough persisted. The patient left the hospital of his own accord on Nov. 27, 1923, before being discharged.

Second Entry.—On July 19, 1924, the patient reentered the hospital because he had a cough and expectorated copious blood-streaked sputum. In February, 1924, in the interval between admissions, he had a cold with some pleurisy pain and expectoration of blood-streaked sputum. He was not able to work for a month. The chronic cough persisted with large amounts of slightly foul-smelling sputum, most of which was raised in the morning.

The results of the physical examination were negative, except for the observation of râles, slight dulness and amphoric breathing at the base of the left lung.

The report on July 2 showed that the patient raised from half a cup to one cupful of sputum daily. Repeated examinations of the sputum failed to show elastic fibers or tuberculosis organisms.

On July 25, bilateral empyema of the maxillary sinuses developed. Drainage was employed.

On August 29, iodized oil was injected into the trachea and x-ray pictures taken. Large bronchiectatic cavities were seen in the lower lobe of the left lung behind the heart (fig. 8 *A*).

Operation.—On Sept. 5, 1924, with the patient under procaine hydrochloride and gas oxygen anesthesia, an incision was made between the sixth and seventh interspaces on the left side extending from the front to the angle of the ribs behind. The lower lobe was partially collapsed. It was small, dark, congested and indurated. It did not expand under positive pressure. The pedicle was mobilized. There were few adhesions attaching the lung to the diaphragm. The separation of the lower lobe from the middle lobe was somewhat more difficult. The pedicle was treated as in the previous case; the lower lobe was removed.

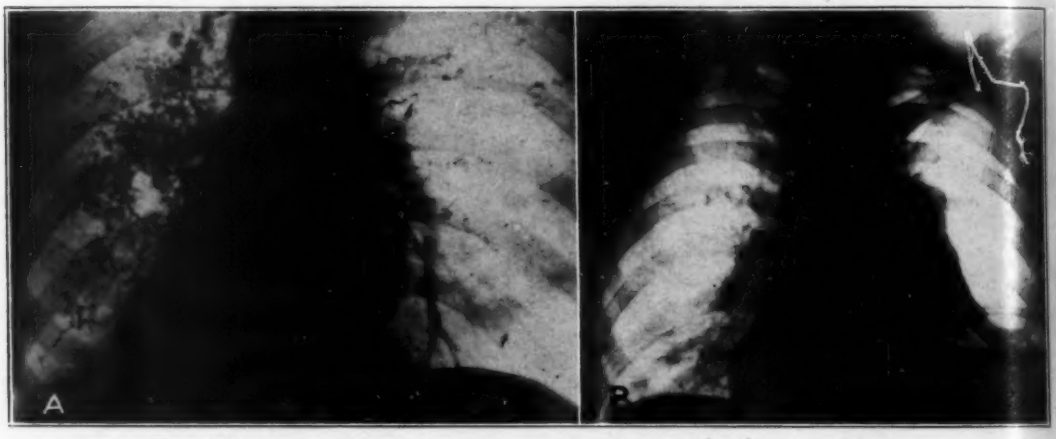

Fig. 8 (case 4).—*A* shows J. M., on Aug. 29, 1924, after an injection of iodized oil 40 per cent, demonstrating multiple cavitations behind the heart (poorly shown in print); *B*, on Sept. 20, 1924, after removal of the lower lobe of the left lung. Imperfect closure of the bronchus at the pedicle permitted complete collapse of the upper lobe as shown in the figure with a pyopneumothorax which complicated the operation.

Following operation, the patient raised a considerable quantity of sputum and was very dyspneic for a few days. This occurred because I was unsuccessful in completely closing off the bronchus. It was impossible to keep the chest free from air by suction, and there was a continual collapse of the upper lobe of the lung (fig. 8 *B*).

On October 4, one month later, the amount of sputum was slight, and the chest wound was healing rapidly. An x-ray report, however, showed a large pneumothorax and the upper lobe entirely collapsed against the spine. The bronchial fistula persisted; fluid constantly collected in the chest, and a rib resection was done.

On Jan. 14, 1925, the left side of the diaphragm reached the level of the ninth rib and the fourth rib in front. There was a small amount of fluid in the left side of the chest. The lung was still collapsed.

On March 27, the patient was apparently well; the wound in the chest was healed; there was no bronchial fistula, and he was afebrile (fig. 9).

After his operation, the patient remained fairly well and was able to do light work.

He was again admitted to the hospital in the winter of 1927 for pneumonia and for care of his sinuses, and he has failed to gain much in weight. The collapsed lung, however, has expanded and completely fills the chest; there is no deformity.

Examination of Specimen.—Gross examination of a specimen weighing 120 Gm. was made on Sept. 6, 1924. The surface was dark purple and showed a few fibrous adhesions over the posterior and diaphragmatic surface. On palpation, it was firm and fairly boggy; there was crepitation only on the edges. There was evidence of interstitial fibrosis, and the dark red color suggested the presence of a large amount of blood. On section, most of this lobe was found to be occupied by large, thick-walled bronchiectatic cavities; these cavities appeared shrunken after fixation, but some were still present and measured from 1 to 1.5 cm. in diameter. There was

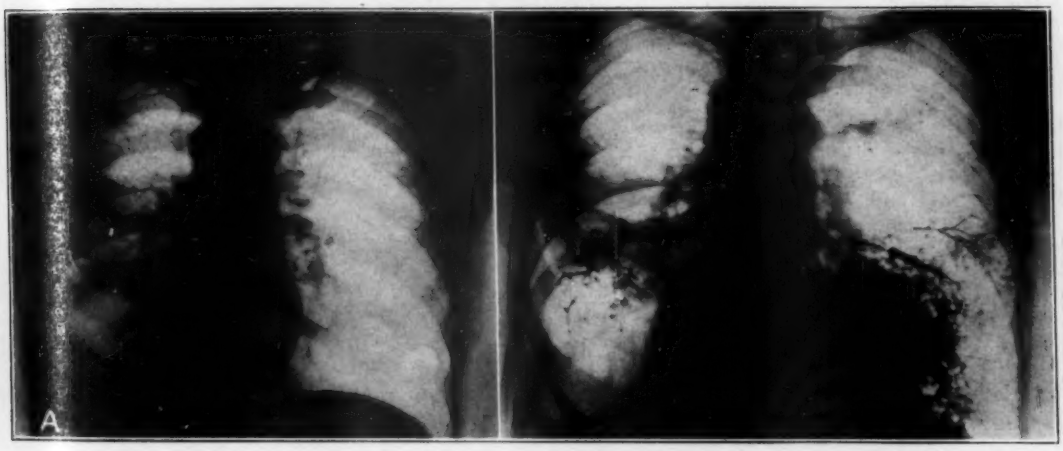

Fig. 9 (case 4).—*A* shows complete expansion of the upper lobe in J. M. on Feb. 16, 1926; *B*, after an injection of iodized oil showing a small cavity at the pedicle. The patient is in good condition. There was no bronchial fistula.

considerable fibrosis immediately around these areas. Dilated blood vessels were also seen on cut section.

Microscopic examination showed the bronchi greatly dilated, with the contour of the epithelium broken in certain areas. In some areas the epithelium was small and flat; in others, it showed hyperplasia with localized thickening, and in still others there was a definite transition to a type resembling stratified squamous epithelium. About these bronchi there was marked infiltration of lymphocytes, with the formation of small germinal follicles in some areas. A moderate number of plasma cells and eosinophils were also seen. The smaller bronchioles throughout the substance of the lung also showed dilatation with compression and irregularity of outline in some. About these bronchi there was marked fibrosis with considerable interstitial fibrosis throughout the substance of the lung. The alveoli were small, showing a considerable degree of atelectasis. The striking feature was the large

amount of blood found in this lobe, both in the dilated capillaries and in the interstitial tissue of the lung. Nothing was seen to suggest tuberculosis in any sections.

A diagnosis of extensive bronchiectasis of the lower lobe of the left lung was made.

CASE 5.—*History.*—J. C., a clerk, aged 22, was admitted to the Southern Pacific Hospital on Sept. 19, 1927, because he had a chronic cough. It was thought that he had tuberculosis. The present illness began following scarlet fever at 4 years of age, when a productive cough developed. He had always raised a mucopurulent sputum. About six months before admission to the hospital, the cough seemed to increase in severity, and the quantity of mucopurulent sputum was greatly increased. There were no night sweats, but the patient said that he had lost 5 pounds (2.3 Kg.) in weight in the last six months. Hemoptysis was not present. As far as the patient knew, he had not been exposed to tuberculosis in his family. The past

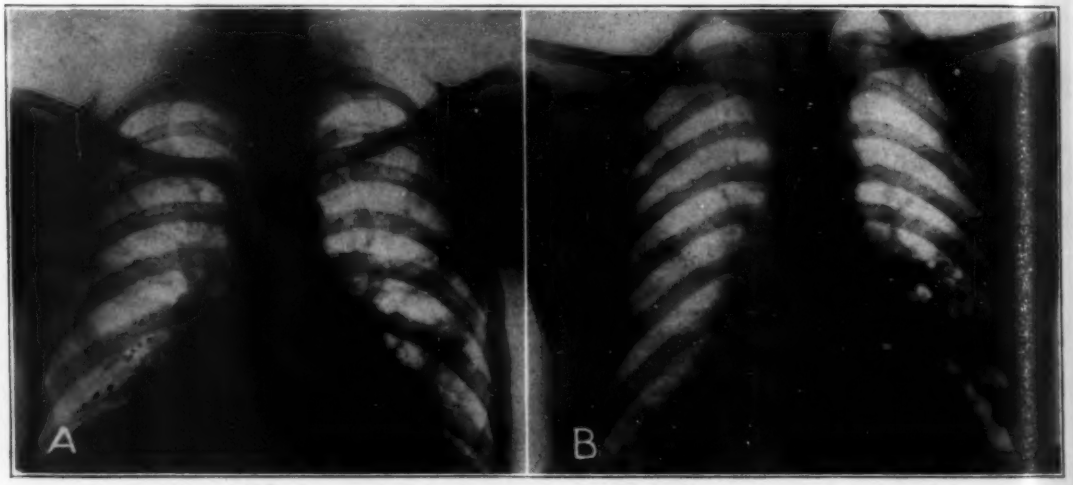

Fig. 10 (case 5).—*A* shows J. C., after an injection of iodized oil. Dilatation of the bronchial tree is seen on the right side. *B* shows cavitations on the left side behind the heart following a similar injection. The left lobe was removed. The patient died from hemorrhage.

history showed that he drank two cups of coffee a day, but did not use tobacco, alcohol or drugs. He was sometimes restless during the night due to the cough. His appetite was good. The bowel movements were always regular. He had occasional frontal headaches and cold. The cough had been present for seventeen years since he had had scarlet fever. There was no dyspnea on exertion or palpation. The gastro-intestinal system was normal. Frequency, nocturia or symptoms of disease of the genito-urinary system were not present. Six months before admission to the hospital, the average weight was 135 pounds (61.2 Kg.). On admission, the weight was 130 pounds (59 Kg.).

Examination.—The chest showed limitation of expansion on both sides with no intercostal bulging. Fremitus was diminished over both lower lobes. There was marked dulness at the lower lobes, especially that of the left lung. The breath sounds were bronchial with numerous coarse râles over both lower lobes. All signs

were more marked in the left lung; otherwise, the results of the physical examination were negative, except for a marked degree of clubbing of the fingers.

X-ray examination showed bronchiectatic dilatation of the lung on both sides, more marked on the left (fig. 10). The patient was sent to Arizona; his condition became somewhat worse, and he returned to the Southern Pacific Hospital for operation.

Operation.—The usual intercostal incision was made on the left side between the seventh and eighth ribs. Scopolamine, 1/200 grain, and 1/8 grain of morphine was injected one and a half hours before operation. Procaine hydrochloride, 0.5 per cent, was given locally. Although the patient was a nervous boy, aged 22, he did not suffer any particular pain, and the operation could be finished without further anesthesia. The diseased lung was somewhat collapsed and thickened. There were a considerable number of adhesions to the diaphragm and in the interlobular septum. These were separated and tied. The pedicle was mobilized; however, it was fairly large and rather thick. Two Wertheim right angle clamps were necessary to encompass it. The pedicle was tied off on the venous side first, and the stump was whipped over with a running suture of chromic. Particular care was taken in closing the bronchus. The patient left the table in good condition, and I had no serious qualms about the result. However, the operation was done in a hospital in which I was unaccustomed to work, and I was not notified when the nurse was unable to withdraw any air or fluid from the chest through the tube. When I saw the patient I was able to start the suction with a little salt solution and found that almost pure blood was coming from the chest cavity. The patient's condition was low from the loss of blood, and reoperation was impossible. He died several hours later. Postmortem examination was not obtained. Through some error in technic, the patient died of hemorrhage. This is the first case I have had in which hemorrhage gave me any concern, and I believe that if I had dilated the lungs before the closure and sealed the pedicle as described this untoward result might have been avoided.

Microscopic Examination.—The specimen consisted of a somewhat flattened mass of lung, probably representing most of the lower lobe of the right lung. It measured 13.5 cm. in length on the outer surface and 15.5 cm. on the inner surface. The upper portion measured 4.5 cm. in breadth and the lower, 8 cm. From the lateral surface toward the hilum, it averaged 3.5 cm. in thickness. The upper two thirds of the lung was crepitant and mottled. This was due to irregular areas of grayish-white parenchyma, varying in size up to 0.5 cm., that were somewhat elevated and represented small areas of emphysema. The intervening parenchyma was a deep reddish blue and showed the presence of small bits of anthracotic pigment. The lower third of the lung on the external surface appeared opaque and grayish white. It was firm and rubbery. The inner margin of the lung represented the surgical surface. A transverse section through the lung on a plane 1 cm. beneath the pleura showed numerous dilated bronchioles, some of which in cross-section measured about 8 mm. in diameter from the outer walls. The lumina varied in size. The walls were greatly thickened by dense circular strands of connective tissue from 2 mm. up to about 4 mm. in thickness. Some of the adjacent bronchioles were bound together by dense strands of connective tissue radiating out into the parenchyma of the lung. As the hilar region was approached, the bronchioles showed an increase in the dilatation of the lumina, and here the walls became more cartilagenous. In the adjacent parenchyma might be seen islands of yellow, slightly elevated, irregular, pinhead foci that suggested large accumulations of fat-laden cells in the alveoli. In between these were delicate, translucent strands of connective tissue. This was especially true in the basilar

portion. Along some of the bronchioles were edematous, slightly enlarged lymph nodes that contained a moderate amount of anthracotic pigment. On microscopic examination, sections taken from the lower lobe showed a moderately thickened pleura made up of compact avascular, acellular connective tissue. In the parenchyma, the bronchioles were prominent. The mucosa of these was thrown into multiple folds. About the bronchioles there was a marked increase of moderately cellular, dense connective tissue. The muscle elements were not distinctly made out. Within this connective tissue was a dense infiltration of lymphocytes and plasma cells. The infiltration in some areas was so heavy as to simulate lymphoid tissue. It extended out into the alveolar walls of the adjacent parenchyma, and a moderate amount of interstitial fibrosis was associated with it. Here and there in the alveoli were foci of organization intermingled with alveoli containing large numbers of fat-laden cells. In many alveoli were accumulations of lymphocyte and plasma cells, which were interspersed with small capillaries from the alveolar walls. Weaving in and out were delicate fibrils of connective tissue.

Sections stained with Mallory's connective tissue stain confirmed the presence of marked peribronchiole and interstitial fibrosis. In addition, delicate blue stained fibrils of connective tissue were seen in the alveolar spaces.

On microscopic examination, it was concluded that the condition was bronchiectasis with interstitial and organizing pneumonia.

CASE 6.—*History.*—I. B., a widow, aged 41, complained of cough with blood-streaked sputum, loss of weight and weakness. According to the family history, the father and mother were living and well. There was no history of disease or tumors. The patient's husband died from tuberculosis. Four children were living and well. The past history showed that the patient had resided in California most of the time; also in the East and middle West. She had had the usual childhood diseases, measles, mumps and whooping cough. She had influenza in 1918, at the age of 31 years.

The patient was apparently well up to September, 1926, and her weight was about 175 pounds (79.4 Kg.). At this time, she developed swelling of the ankles, most marked in the morning. The following months, she became languorous. Her endurance began to fail; her appetite became poor; she could not sleep, and constant dull pain developed in the right thigh. She had a cough productive of some blood-streaked sputum. There was some palpitation and breathlessness, especially on exertion. By the end of December, the cough had become pronounced, the patient had lost some weight and appeared ill. Early in January, she attended the University of California clinic. Her weight was 147 pounds (66.7 Kg.). Examination of the chest revealed little of note; there were some bronchial breath sounds on the right side without râles. The fingers and toes were markedly clubbed. X-ray examination showed in the lower right side of the chest a shadow which had the appearance of sequelae to a respiratory infection and possible malignancy. During the early part of January, she was in an automobile accident, being caught behind the steering wheel. She was taken from the automobile apparently unhurt and started home on a street car. She became confused in transferring; she was disturbed and cried, and could not remember the way home. During the same month, while in her car, she had a generalized tonic convulsion; the pupils dilated with rotatory nystagmus, and there was some foaming at the mouth. During the next hour, she had five or six similar attacks and was taken to the University of California Hospital in coma. There was a questionable bilateral Babinski sign, and left rotary nystagmus. Memory of the convulsive attacks was absent, and memory of the preceding weeks was hazy. There were a few hilum râles. An

electrocardiogram indicated slight myocardial changes. The Wassermann reactions of the blood and the spinal fluid were negative.

It had been noted by the relatives that since the time of the accident there were, besides the convulsions, definite changes of personality characterized by irritability, obstinacy, lack of cooperation and diminished power of memory and reasoning. After three days, she recovered sufficiently to return home.

The following month attacks of hemoptysis began, associated with persistent aching pain over the lower right side of the chest, posteriorly. This was aggravated by deep breathing and coughing. Chest signs at this time remained unchanged. During the same period, she had difficulty in vision and frontal headaches occurred. Examination of her eyes revealed 20/40 vision of the right eye, which was corrected with glasses.

Cough, hemoptisis and expectoration increased progressively up to June, during which time she lost 20 pounds (9 Kg.) in weight. In July, the hemoptysis stopped and the amount of sputum decreased. On August 11, bronchoscopy was performed; the main bronchus of the right lung appeared reddened and the left was narrowed by a tumor which was growing over. No section was taken. Five days following bronchoscopy, the patient had another convulsive seizure and was taken to the San Francisco Hospital on August 18. She was extremely weak and irritable and her memory was poor; the temperature was 103 F.; septic; the pulse rate, 120; respiration, 35 and hemoglobin 90 per cent. The blood count showed: red blood cells, 4,800,000; white cells, 6,250; polymorphonuclears, 70 per cent. Dark yellow fluid was removed from the right side of the chest. The chest signs gave evidence of atelectasis of the middle and lower lobes of the right lung. Both disks showed some choking at this time. The sputum decreased to about 30 cc. daily and was without blood or odor.

A large series of x-ray pictures were taken; the changes which were noted were of extreme interest and led one to believe that the patient was suffering from a malignant condition in the bronchus. On three bronchoscopic examinations, I was not able to prove this absolutely.

The first x-ray picture taken in January showed only a slight shading in the middle of the lower lobe. By April (fig. 11 *A*), this had increased to a moderate extent. In July (fig. 11 *B*), there was atelectasis of the lower lobe, and by August (fig. 12 *A*), the middle lobe was also involved. Because of the lack of a better diagnosis, an exploratory operation was decided on for diagnostic purposes, although the patient was in a miserable condition.

Operation.—On Sept. 9, 1927, operation was performed at the San Francisco Hospital, University of California service. Preliminary injection of $\frac{1}{200}$ grain of scopolamine and $\frac{1}{6}$ grain of morphine was given one and one-half hours previous to operation and a second dose of $\frac{1}{400}$ grain of scopolamine and $\frac{1}{8}$ grain of morphine a half hour before operation. Procaine hydrochloride, 0.5 per cent, was injected. The operation was performed without general anesthesia. The usual intercostal incision was made. The upper and middle lobes were hard, firm, rather mottled and not crepitant. There were a few adhesions to the diaphragm and also between the middle and upper lobe. As the patient's condition was good, I decided to remove the diseased lobes, and the procedure was carried out as described in this article.

The patient made a good recovery from the operation. There was practically no shock, and the next morning she was reading the paper. Suction was kept up every two hours day and night for a period of five days. At the end of three days, an x-ray picture showed (fig. 12 *B*) the upper lobe expanded and only a small area left where the lower lobe had been removed. The patient was out of bed in

Fig. 11 (case 6).—*A* shows I. B., on April 6, 1927, with slight changes in the lower lobe of the right lung. The results of the examination were negative; *B* shows the same patient on July 12, 1927. Atelectasis (massive collapse) of the lower lobe of the right lung is seen and the heart is drawn to the affected side.

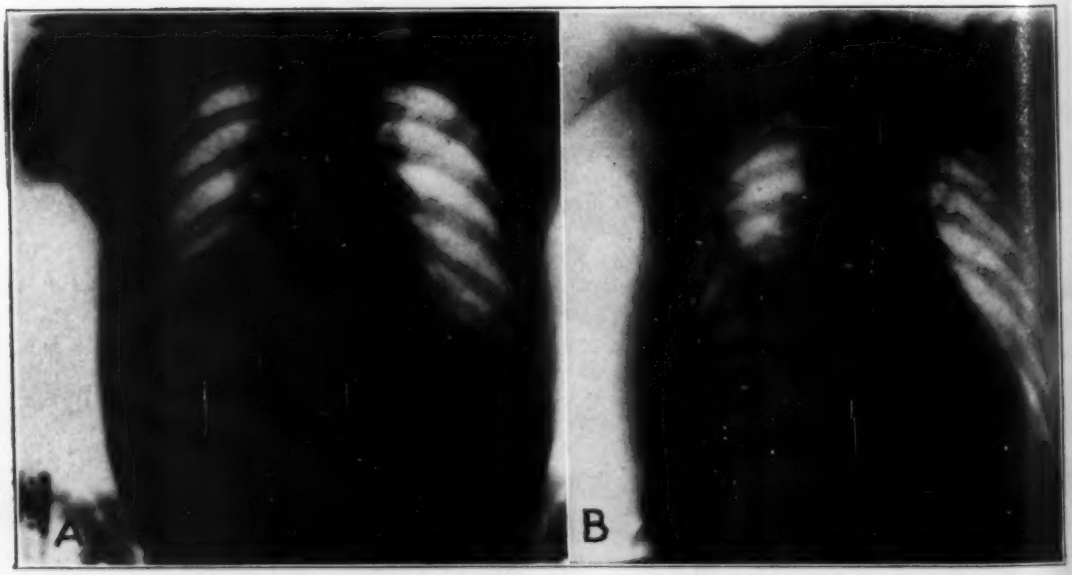

Fig. 12 (case 6).—*A* shows I. B., on Aug. 10, 1927. The lower and middle lobes are atelectatic. *B* shows the same patient on Sept. 19, 1927. The lower and middle lobes of the right lung have been removed. The x-ray plate was taken three days after operation showing the expansion of the upper lobe and a small empyema cavity left below.

two weeks. At the end of three weeks, resection of the rib was made on account of a slight rise in temperature. The operative recovery was ideal. Examination of the removed lobes did not show any evidence of carcinoma, although I felt that some must still be present in the hilum and the mediastinum, but x-ray examination even at this time gave no clue. However, the malignant condition grew worse, and she finally became bedridden.

The headaches and the choked disks disappeared, although the patient was always mentally unbalanced. The first definite evidence of a malignant condition was obtained through an operation performed on her right ankle, which had become very painful. There were evidences of osteo-arthropathy of both knees and feet, and an x-ray picture showed also destruction of the bone. Specimens removed showed adenocarcinoma. The patient died on June 6, 1928.

Microscopic Examination.—A specimen examined on Sept. 7, 1927, consisted of the lower lobe of the right lung which measured 11 cm. in the vertical plane, 5 cm. in the anterior posterior and 8 cm. in the lateral plane; and another specimen, apparently the middle lobe, measuring 8 by 7 by 4 cm. A few small fibrous tags were seen on the outer surfaces of both of these lobes, especially on the diaphragmatic surface of the lower lobe. In general, the lateral surface was opaque, yellowish-white with a few small scattered foci of anthracotic pigmentation. The tissue was firm and rubbery and sank in the formaldehyde solution. A vertical section made through the transverse plane showed a remarkable picture. The upper half of the lower lobe, about the periphery, showed that the alveoli were obliterated by a diffuse process made up of minute finely granular, yellowish foci which here and there were divided off by translucent strands of connective tissue. In the lower part of the lobe there was a conglomeration of this material to form larger foci measuring from a few millimeters to 0.5 cm. They appeared elevated, circumscribed and somewhat irregular, and about them were strands of fibrous tissue; the appearance was not unlike that of cirrhosis of the liver. The bronchi were prominent, but not dilated and were surrounded by a slight increase in connective tissue. They contained a purulent exudate. The mucous membranes appeared hyperemic. There was no evidence of abscess formation. The specimen did not contain the bronchial lymph nodes. The parenchyma of the middle lobe showed essentially the same picture.

Microscopic examination of several sections showed the pleura in general to be of normal thickness, although here and there the capillaries were dilated, causing it to appear hyperemic. The underlying parenchyma was of a variable appearance. Areas of from twenty to thirty adjacent alveoli showed thin walls, but contained precipitated serum, a few polymorphonuclear leukocytes, occasional strands of fibrin and a large number of large, pale, fat-containing cells, as demonstrated by the scharlach r stain. The accumulation of such cells gave the yellow granular appearance seen in the gross specimen. Adjacent to such areas were larger, usually circumscribed foci, about the periphery of which the alveoli were filled with a varying number of lymphocytes and delicate strands of cellular fibrous tissue that contained small, thin-walled capillaries. The alveolar walls themselves showed lymphocytic and plasma cell infiltration and strands of fibrosis. The connective tissue was excellently demonstrated by Mallory's connective tissue stain, and in the alveoli, especially toward the hilar portion of the lung, showed marked organization produced by sheetlike strands of vascular fibrous tissue. There was also an increase in the peribronchial connective tissue. Many of the small bronchioles contained an exudate of polymorphonuclear leukocytes. The section did not show any evidence of malignancy.

Following microscopic examination, it was concluded that the condition was subacute organizing pneumonia and interstitial pneumonia.

Postmortem Examination.—On June 6, 1928, the anatomic diagnosis was: primary carcinoma of the right lung of the type of papillary adenocarcinoma, arising in about the first division of the primary bronchus and extending into the hilar nodes, pericardium, about the inferior vena cava, into the space left by lobectomy on the lower lobe of the right lung, between the ribs on the right, into the sinus resulting from the operation for empyema, into the right side of the diaphragm and into the mediastinum; there were metastases to the left lung, retroperitoneal nodes, culdesac, liver, suprarenals, ovary, dura, bones of the skull, brain and skin of the ankle. The lower and middle lobes of the right lung were absent as a result of lobectomy.

Postmortem examination also showed: block of the bronchi to the upper lobe with abscess formation and chronic organizing pneumonia; empyema sinus in the right side of the wall of the chest entering an abscess cavity in the tumor mass, the sinus being lined by the tumor mass; obliteration of the right pleural cavity by the tumor; left pleural effusion (1,000 cc. of blood-tinged fluid) with compensatory atelectasis of the left lung; ascites, 300 cc. of clear fluid; dilatation and hypertrophy of the right side of the heart; chronic passive congestion of the viscera; moderate atheroma of the heart valves and aorta and coronaries; subacute vaginitis, cervicitis and cystitis; cholelithiasis (three large cholesterol stones and about 100 small pigment stones); polyp of the duodenum; acute splenic tumor; block of the thoracic duct in the mediastinum with edema of the retroperitoneal tissues; anemia; emaciation; healed operative wounds, lobectomy and removal of metastasis in the left lower leg.

Postmortem Microscopic Examination.—The slides from the heart showed some edema and cloudy swelling of the muscle. The valve sections were atheromatous. The slides from the aorta were atheromatous. The liver showed congestion. There was fat phanerosis and focal necrosis. There were tumor nodules showing epithelial cells making up an adenomatous growth with marked papillary formation. There were some necrosis and marked fibrosis in the tumor. The slides from the spleen showed acute splenic tumor. There was congestion. The left lung showed alternating atelectasis with emphysema. There was a small tumor nodule under the pleura. There was some congestion. The right lung showed a massive invasion of an adenocarcinoma springing from the bronchus. The tumor was composed of epithelial cells without much arrangement in the lung. There was marked sclerosis with only a little necrosis. The upper lobe showed abscesses and organizing pneumonia. The bronchus showed the tumor in the peribronchial lymphatics, but an actual site of origin could not be found. The adjacent nodes were filled with tumor. The tumor had invaded the nodes as far down as the retroperitoneal nodes about the origin of the renal arteries. Slides from the culdesac showed tumor. The slides from the duodenum showed a polyp. There was some chronic infiltration with round cells. The pancreas showed slight fibrosis. The slides from the suprarenal showed cloudy swelling with metastases of tumor in them. The uterus showed a cystic endometrium. There were a few myomas in the walls. There was a chronic cervicitis with many nabothian cysts. The ovaries showed many corpora albicans and one area in which there was a bit of tumor. The slides from the vagina showed blebs, ulcers and inflammation. There were thrombosed vessels in the tissue back of the vagina. The bladder showed moderate inflammation. The cortex of the brain appeared normal. Over the inferior surface of the left frontal lobe, there was a tumor on the inner surface of the dura and rather firmly attached to the dura. The surface of the tumor adjacent to the brain was slightly adherent. The tumor was grayish white, very nodular and measured 6 by 4 cm. in length and width and about 1.5 cm. in thickness.

All the gyri over the anterior posterior of the inferior surface and frontal lobe as well as the anterior surface of the first, second and third frontal convolutions showed evidence of pressure and apparent atrophy. Sections through the brain stem, cerebellum and right cerebral hemisphere were negative. Sections through the anterior end of the left frontal lobe showed a cavity in the inferior portion of the frontal lobe with softening around it. This cavity was about 3 cm. in length, 3 cm. in width and 1 cm. in height. It extended to within 1 mm. of the inferior surface of the frontal lobe, the inferior wall being merely a thin layer of gray matter. The cavity was lined with a brownish-gray substance. Sections posterior to the tip of the frontal lobe did not show a pathologic process.

The anatomic diagnosis was: metastatic carcinoma of the dura over the left frontal lobe; atrophy of the convolutions of the inferior surface of the left frontal lobe and of the anterior portion of the first, second and third frontal lobes.

There was a cyst in the base of the left frontal lobe. Sections from the wall of the cyst in this lobe showed numerous carcinomatous cells. There were large collections of both these and numerous seedings. In some areas the cells had an irregular arrangement, but in other areas there was distinct acinar or adenomatous arrangement. Some of the carcinoma cells were exceedingly large. In this area there was also softening with disintegration of the brain tissue and gliosis. The leptomeninges in this area also contained carcinomatous cells. A section from the nodule of the dura showed extensive infiltration of the dura with carcinoma and a proliferation of the connective tissue. The diagnosis was metastatic carcinoma of the dura and metastatic carcinoma in the inferior portion of the left frontal lobe with softening and cavity formation.

EXPERIMENTAL PRODUCTION OF ABSCESS OF THE LUNG*

A. V. S. LAMBERT, M.D.
AND
CARNES WEEKS, M.D.
NEW YORK

The literature on pulmonary suppuration and pulmonary gangrene up to 1920, although voluminous, is extremely indefinite as to the rôle which anaerobic organisms might play in the etiology of these diseases. Since that time, an increasing number of articles have appeared in which the authors mention the possible etiologic importance of these organisms as the causative agents in these conditions. Since 1921, several authors have mentioned the constant presence of anaerobes in suppuration of the lungs; in one series of operative cases, they were the only organisms found in eight of ten instances in which pus was removed from the abscess cavity.

Although heated controversies have been waged, especially in France, and voluminous theoretical discussions have been reported about the significance of the various groups of anaerobes which are found in the sputum of patients suffering from these diseases, it is surprising how little experimental work has been done, and in our studies of the literature we have found that this work is confined almost wholly to workers in the United States.

Considerable work has been done, however, on the action of anaerobes in the subcutaneous tissues and by direct inoculation into the pleural cavities.

In 1923, Kline [1] produced "pleuropulmonary gangrene" in a rabbit by "intrabronchial injection" of material from a carious tooth containing spirochetes and fusiform bacilli. The pleural exudate from this animal caused subcutaneous suppuration when injected into other animals, and spirochetes and fusiform bacilli were recovered from these subcutaneous abscesses.

In 1924, Lambert [2] injected mixed cultures of pure anaerobes into the ear veins of rabbits, and after repeated failures, caused in one animal an abscess of the lung following a suppurative thrombophlebitis arising at the site of injection. The same organisms were recovered from the pulmonary abscess.

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* From the Department of Surgical Research, Cornell University Medical College, New York.

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2. Lambert and Miller: Abscess of the Lung, Arch. Surg. **8**:446 (Jan.) 1924.

In 1925, Ribierre and Kermorgant [3] produced hemorrhagic bronchitis and conjestion of the lung by intracheal inoculation of 1 cc. of a culture of spirochetes and bacilli in horse serum. They do not give any more definite description of the organisms. With the bacillus in pure culture, they obtained negative results. They were not able to isolate the spirochetes in pure culture.

In 1926, Cutler and Schlueter [4] produced pulmonary abscesses by introducing into the jugular vein an embolus composed of a portion of the femoral vein containing a small piece of lead, blood, two silk ligatures and various aerobic organisms.

In 1927, Smith [5] reported that by using washed sputum from patients with pulmonary suppuration and also by using scrapings from persons suffering from pyorrhea, he produced by intratracheal injection "all types of acute and chronic fusospirochetal disease" in the lungs of mice, guinea-pigs and rabbits.

In the same year, Ghiron and Maddalena [6] produced abscesses with a segment of vein, containing gastric and intestinal contents, introduced into the jugular vein. The organisms recovered were aerobic.

In 1928, Crowe and Scarff [7] reported that they produced pulmonary abscesses in dogs by implanting in a bronchus a pledget of cotton soaked in pyorrheal scrapings and also by producing a frontal sinusitis in other dogs by implanting a similar pledget (of cotton), also soaked in pyorrheal scrapings, in the frontal sinus. It is important to note that after working two years they reported failures so long as they employed aerobes, and obtained abscesses only after they used anaerobes. They demonstrated the presence of spirochetes by dark-field examination, but did not report any cultures.

In 1928, Allen [8] introduced through a catheter into the bronchi of dogs and rabbits warm sputum containing cocci, fusiform bacilli and spirochetes obtained from a case of abscess of the lung, and produced multiple abscesses of the lung; the incidence was higher when the bronchus was coincidently ligated. When the pus had been allowed to cool, he obtained negative results in mice. He reported having frequently caused abscesses of the lungs by emboli, but does not give further details.

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8. Allen, D. S.: Etiology of Abscess of the Lung, Arch. Surg. **16**:179 (Jan.) 1928.

Smith [9] isolated in pure culture the various anaerobes: *Spirochaeta microdentium* and *macrodentium,* two types of fusiform bacilli, and four strains of vibrios, and also three anaerobic cocci—one hemolytic, two nonhemolytic and two viridans type. He reported being unable to produce suppuration of the lung by intratracheal injection of pure cultures of spirochetes.

Without entering the controversy as to whether abscesses of the lung are of aspiratory or embolic origin, believing as we do that they may occur in both ways, we have attempted to study the question from the purely bacterial phase.

We have employed the embolic method believing that it is freer of possible error than the aspiratory where all infective agents must pass through the buccal cavity swarming with countless micro-organisms which might act as contaminants.

The method we have employed is to fill with blood a hypodermic syringe and add to this the infective agents, allow a firm clot to form and introduce this into the jugular vein. We thus avoid the possible objection that the presence of a foreign body—vein, silk or lead—may be the determining factor. We have employed as infective agents washed sputum from a case of abscess of the lungs, cultures of the same sputum, pus obtained at operation from the abscess cavity, cultures of this pus and cultures of pus obtained at autopsy from dogs in which we had caused abscess of the lungs. The time which elapsed between the recovery of this material and its introduction into the animals varied considerably, from a few minutes in the case of the sputum to as long as two hours with the pus from the abscess cavities, but the material was kept at body temperature during this period in every instance.

The organisms present in the material used and in the pus recovered from the dogs' abscesses have been: (1) spirochetes, (2) fusiform bacilli, (3) a large gram-positive bacillus, (4) a medium sized gram-negative bacillus, (5) a small gram-negative bacillus and (6) a gram-positive coccus of the viridans type. We have not used pure cultures of any of these organisms.

We have obtained single abscesses in the lungs of dogs with fresh washed sputum and with pus removed from abscess cavities at the time of operation. In one dog, we obtained two abscesses. One was situated in the lower lobe of the right lung and the other in the lower lobe of the left lung; this we interpreted as a breaking up of the clot in the heart into two separate emboli. It is interesting to note in this connection that when Cutler used a clot embolus containing aerobes he reported the production of diffuse pneumonitis without abscess formation. Smith and Allen, employing anaerobes intratracheally, whether they were anaerobes alone is not certain, produced multiple suppurative lesions.

9. Smith, D. T.: Am. Rev. Tuberc. **16**:584, 1927.

It is interesting that when washed sputum, containing both aerobes and anaerobes, was used, the abscess produced by it contained only anaerobes, the aerobes having disappeared.

We failed to produce abscesses employing cultures of anaerobic organisms four, six and eight days old. These cultures did not contain spirochetes. We did, however, produce an abscess in one case with a culture two days old obtained from the pus of a dog's abscess experimentally produced. This culture contained spirochetes. We have no solution to offer for our inability to produce abscesses with cultures over two days old.

The time of incidence of the abscesses in the dogs varied from three to seven days with an average of six days. This time is difficult to determine with great accuracy, but we have considered the following factors: the animal's rise of temperature, his general condition and the x-ray evidence. The latter is not always easy to interpret. The duration of the abscesses varied from four to forty-six days. The death of the animals not killed for study was usually due to pulmonary hemorrhage or acute empyema following rupture of the abscess.

The situation of the lesions was usually peripheral and in the lower lobes, the cavity of the abscess containing a green, foul-smelling exudate. Sections of the tissues showed a central necrotic area with a surrounding zone containing spirochetes.

CONCLUSIONS

1. Many abscesses which contain only anaerobes occur in human beings.

2. The anaerobes associated with abscess of the lung in man are pathogenic for dogs.

3. Single discrete pulmonary abscesses may be caused in dogs by employing anaerobes alone in an embolus of blood clot.

4. The anaerobes are not simply secondary invaders in abscesses of the lungs, but can and do give rise to suppuration in the lungs.

5. After introduction of an embolus containing aerobes and anaerobes, the aerobes may disappear and only the anaerobes remain.

RECURRING HEMORRHAGE IN CHRONIC SUPPURATIVE CONDITIONS OF THE LUNG

TREATMENT BY LIGATION OF THE PULMONARY ARTERY

REPORT OF TWO CASES *

HOWARD L. BEYE, M.D.
IOWA CITY

Ligation of the pulmonary artery as a means of controlling the disease in cases of chronic infections of the lung, tuberculous or otherwise, has been done by Sauerbruch, Meyer, Lilienthal and others. The results have not been sufficiently striking to establish the operation as one of choice. Apparently in most instances palliation only was obtained, and better results are obtained by extrapleural thoracoplasty.

Ligation of the pulmonary artery has been suggested by several writers as a step preliminary to lobectomy in cases of bronchiectasis of a single lobe or of neoplasm.

In the following two cases, ligation of the branch of the pulmonary artery to the lower lobe of the right lung was performed in an attempt to control the symptom of hemoptysis. In each case partial thoracoplasty had been performed, together with phrenic neurectomy, and in each the symptom of pulmonary hemorrhage persisted to such a degree, in spite of surgical collapse, that a fatal termination seemed inevitable.

REPORT OF CASES

CASE 1.—*History.*—Mrs. Robbins, aged 36, entered the University Hospital on July 20, 1926. Two years prior to this, a cesarean section was performed under ether anesthesia; pneumonia developed seven days later and was followed by a suppuration of the lung. For one month, she coughed a great deal and raised foul sputum. In December, 1924, an abscess was drained, following which she made considerable improvement. Drainage through the wound gradually decreased, although it never stopped completely. Cough persisted, yellowish and foul and frequently blood-streaked sputum being expectorated, averaging one-half cupful daily.

Examination.—The patient was a moderately obese woman, with pasty complexion. The results of the general examination were negative, except for the lungs. On the right side posteriorly, there was decreased resonance from the level of the scapular spine to the base posteriorly and to the midaxillary line. Over this area, the breath sounds were tubular, and the spoken and whispered voice sounds were freely transmitted. There was a probe-sized draining sinus at the level of the scapular angle. The hemoglobin content was 85 per cent; the red cells totalled 4,800,000, and the leukocytes, 8,600.

Roentgen-ray examination of the chest disclosed a dense shadow on the right from the level of the seventh rib posteriorly to the diaphragm. Injection over the

* From the Department of Surgery, College of Medicine, University of Iowa.

base of the tongue of 20 cc. of iodized oil—40 per cent outlined an irregular cavity of fair size and several smaller cavities in the lower lobe of the right lung.

A diagnosis of chronic pulmonary suppuration with multiple bronchiectatic cavities was made.

Treatment and Course.—With the patient under procaine hydrochloride anesthesia, several centimeters of the right phrenic nerve were avulsed. Following this procedure, she made a good recovery and returned home for a few weeks. While there she had an acute exacerbation of the pulmonary infection, which was followed by a moderately severe hemoptysis. A few days later she had two more attacks of bleeding and brought up about a cupful of blood with each attack. She again entered the hospital, and while under observation had two slight hemorrhages.

Graded thoracoplasty was performed with subperiosteal resection of the major portions of the third to the tenth ribs, inclusive. A considerable degree of collapse of the wall of the chest was obtained.

The patient did not show much subjective or objective improvement following this procedure. The sputum varied from 30 to 150 cc. in twenty-four hours, and two weeks after the third stage of the thoracoplasty, she raised 50 cc. of bright blood. Her temperature ranged between normal and 99.5 F. She was discharged on the nineteenth day following the completion of the surgical collapse. During the following six weeks, she had repeated hemorrhages of from 30 to 50 cc.; finally, she had a severe hemorrhage during which she raised over 500 cc. of bright blood.

She entered the hospital again on Feb. 13, 1927, in poor condition. The hemoglobin was 55 per cent (Sahli); the red blood cells, 3,300,000; the hematocrit, 25 per cent, and the white blood cells, 10,200.

Second Operation.—On February 17, under ethylene anesthesia, an 8 inch (20.32 cm.) incision was made in the third right interspace and the costal cartilage of the third and fourth ribs divided just lateral to the mammary artery. There was considerable decrease in the size of the thoracic cavity owing to the thoracoplasty, and the diaphragm lay at the level of the lower margin of the fourth rib. The lower lobe was tightly adherent to the posterior wall of the chest and to the lower half of the middle lobe in its posterior portion. Blunt dissection was made mesially between the middle and lower lobes. This produced moderately profuse bleeding, which was controlled by moist packs. It soon ceased. A branch of the pulmonary artery to the lower lobe was recognized by its pulsation. Dissection was carried to this point and the artery found to be the lateral branch. It was about one-half the size of an ordinary lead pencil. Further dissection mesially and upward exposed the mesial branch to the lower lobe. This was somewhat larger than the lateral. The branch of the pulmonary vein lay mesial to the branches of the artery and was well out of the immediate field of attack. In exposing the lateral branch upward to find its bifurcation with the mesial, the lateral branch to the middle pulmonary lobe was exposed. The branches to the lower lobe were ligated with number 3 plain catgut. In addition, the isolated branch (lateral) of the middle lobe was also ligated. This was done because of the dense adhesions between the middle and lower lobes, suggesting the possibility of extension of the pathologic process between the two lobes posteriorly. The divided costal cartilages were sutured, and the incision was closed without drainage.

The patient had little cardiac or respiratory disturbance until after ligation of the branches. Soon after this, the respirations became shallow and somewhat gasping. Oxygen was given for several minutes with marked benefit. At the conclusion of the operation, the patient's condition was good. The pulse rate was 140, and the quality was satisfactory.

Postoperative Course.—For the first six days, the condition was fairly satisfactory. Extensive subcutaneous emphysema developed. On the second day, aspiration of the chest resulted in a slightly cloudy fluid under a positive pressure of 1 cm. of water. Air was withdrawn until a 3 cm. negative pressure was obtained. This was repeated on the fourth and on the tenth day. On each aspiration, the fluid was sterile to culture. Transfusion of 500 cc. of blood was performed on the sixth day without reaction. This seemed to improve the patient's condition definitely. She was brighter, and the pulse rate became slower and of better quality.

On the seventh day, the temperature rose abruptly to 102.4 F., and signs of a pneumonic process began to appear over the entire right side, but especially over the middle lobe. The leukocytes totaled 8,100. The respiratory effort became labored, and the patient gradually became worse, and died on the twelfth day after operation. From the eighth day until her death she complained of some headache, but there was nothing more to suggest a possible intracranial infection. Autopsy was not permitted.

Comment.—Following phrenic neurectomy and thoracoplasty, the patient's course was unsatisfactory, especially because of increasing hemoptysis, and the ultimate prognosis was grave. It was felt, therefore, that an attempt to control the symptom of pulmonary hemorrhage by ligation of the branch of the pulmonary artery to the diseased lobe was justified. When the lung was exposed, the posterior inferior portion of the middle lobe seemed to be involved as well as the lower lobe, which led to ligation of the branch which supplied this segment of lung. It is possible that the middle lobe was not grossly involved and that infarction followed the disturbance of its blood supply and was responsible for, or greatly contributed to, the fatal outcome.

Case 2.—*History.*—Mrs. Arthand, aged 25, entered the University Hospital on Oct. 18, 1926. In the winter of 1921, the patient had an attack of pain in the right side of the chest, associated with fever and sputum. A residual cough persisted. Each winter she had recurring attacks of pneumonia and her general health gradually failed. In the spring of 1926 she had a chill, high fever and severe cough, which suddenly produced a cupful of foul sputum. Following an attempted aspiration of an abscess of the lung, she raised some bright blood. She was sent to a sanitarium for tuberculosis, where several attempts to locate an abscess by aspiration were unsuccessful. She continued to raise considerable sputum which was frequently blood streaked.

She was referred to the University Hospital for lung mapping and bronchoscopic examination. These examinations demonstrated branching bronchiectatic cavities in the lower lobe of the right lung with marked fibrosis of the lobe from the level of the eighth rib posteriorly to the diaphragm. The outline of the latter could not be distinguished from the dense lung. Physical examination of the chest disclosed dulness over this lobe posteriorly with distant bronchovesicular breath sounds and coarse râles. There were 4,100,000 red blood cells, 13,200 leukocytes, and the hemoglobin was estimated at 75 per cent.

Course.—While under observation during November, the patient had a febrile course ranging between normal and 100.2 F. At the time of her menstrual period she began to cough up blood, and for five days she raised large amounts, both free and mixed, with tenacious sputum.

During December, at the menstrual period, she again began to have hemoptysis, and for about six days she raised blood day and night. On one occasion, she raised about 200 cc. Following the menstrual period, hemoptysis decreased in quantity, but on frequent occasions the sputum contained bright blood. There were 20,000 leukocytes and 5,000,000 red blood cells. Profuse bleeding from the lung recurred

during the January menstrual period and was more marked than in the previous two months. The red cells decreased to 3,600,000.

Operation.—Right phrenic neurectomy was performed on Jan. 12, 1927, under infiltration with procaine hydrochloride. The following day, 60 cc. of bloody sputum was raised and 240 cc. the next.

Second Operation.—On January 19, extrapleural thoracoplasty was performed under ethylene anesthesia. Resection through a posterior incision of segments of the sixth to the eleventh ribs inclusive (total, 63.5 cm.) was done.

Course.—Following these two procedures the patient made a satisfactory recovery, but continued to raise blood in small amounts until shortly before the February menstrual period, when hemoptysis again became alarming. The patient was beginning to lose ground rapidly, and it was felt that continuation of the bleeding would inevitably prove fatal.

Third Operation.—On February 15, under ethylene anesthesia, a 7 inch (17.78 cm.) incision was made in the third interspace anteriorly. The costal cartilage of the third rib was divided just lateral to the internal mammary artery, and the rib retracted upward. The lung was adherent to the parietal pleura by adhesions which were readily separated.

The upper half of the lower lobe, the middle lobe and the lower portion of the upper lobe were exposed. The lower lobe was purplish gray, very firm and had no tendency to collapse. It was firmly adherent to the wall of the chest.

No definite line of cleavage could be made out between the solid lower lobe and the soft compressible middle lobe. By gentle retraction of the lower lobe and dissecting upward and inward a line of cleavage was forced. This produced moderate bleeding. A branch of the pulmonary artery to the lower lobe was located by its pulsation and isolated. It was about three-quarters the size of an ordinary lead pencil. It proved to be the mesial division of the branch. Dissection upward isolated the lateral division, about one-half the size of the mesial, and the two were traced upward to their junction. Double ligation was made of the main branch just above the bifurcation. This did not produce any change in the patient's condition and no alteration in the lobe.

Because of some continuation of oozing from the wound made in dissecting the middle lobe from the lower, it was felt best to use an iodoform gauze strip for pack. The third rib was replaced and a mattress suture used to hold the cartilage in position. Usual closure of the soft tissues followed. There was little general reaction throughout, and the patient returned to her room in excellent condition, with a pulse rate of 120.

Course.—Convalescence was uneventful. On the third postoperative day, the menses appeared. For the first period in five months this was not accompanied by profuse hemoptysis. During the twenty-four hours, the sputum measured 45 cc.; it was thick and gray and contained faint streaks of blood. It never showed more than a pink tinge until the March menstrual period, when there was again some bright blood and clots, but relatively slight in amount.

The patient continued to progress satisfactorily, except for a feeling of tightness in the chest on the right side, until July when there was a moderate hemoptysis at the menstrual period.

Ten months after operation, physical and roentgenologic examination of the chest did not show any appreciable change.

In a recent report, over a year since the ligation, the patient said that she occasionally has some blood-streaked sputum but no severe bleeding. The amount of the sputum has not been materially changed. She has gained 20 pounds (9 Kg.) in weight.

Comment.—The control of the alarming symptom of hemoptysis by ligation of the branch of the pulmonary artery justified the procedure. Otherwise, however, there has not been any marked improvement in the disease and lobectomy should be given serious consideration.

Hemoptysis is a frequent symptom in pulmonary suppuration, but is usually small in amount and of no great moment. If it recurs frequently or in increasing amounts, and especially if associated with a septic course, the prognosis is grave. It is in this type of case that ligation of the pulmonary artery should be given consideration, for two reasons: (1) the pulmonary bleeding may be controlled, and (2) the course of the pathologic process may be favorably influenced. Rarely would one be justified in performing such ligation without first attempting to control the alarming hemoptysis by artificial pneumothorax or extrapleural thoracoplasty.

Successful control of hemoptysis by ligation of the pulmonary artery will depend on the source of the bleeding, and this cannot be determined beforehand. It is reasonable to expect that in certain cases of pulmonary suppuration considerable disturbance takes place in the pulmonary or bronchial circulation, or both, of an affected lobe. The initial infection may have been via the pulmonary artery or the bronchial artery, with resultant thrombosis and infarction. Collateral circulation between the bronchial and pulmonary systems may become extensively developed. Collateral circulation between the pulmonary circulatory system and the general circulatory system may develop by way of vessels through adherent pleural surfaces.

If the hemorrhage is coming from one of the branches of the pulmonary artery, as is commonly the case, the operation should succeed if the patient survives the procedure. If the bleeding is from the venous plexus or pulmonary vein, then some degree of control would be reasonably expected; if from the bronchial artery, bleeding may not be affected or may be made worse. Similarly, if collateral circulation through the pleura is firmly established, bleeding may continue through such radicals after ligation of the pulmonary artery.

If hemoptysis is controlled by ligation of the pulmonary artery, recurrence of this symptom may take place because of the subsequent development of collateral circulation through the bronchial vessel. Schlaepfer has shown in animal experimentation that such collateral circulation is retarded by paralyzing the diaphragm, and, therefore, phrenic neurectomy should also be done either before or at the time the pulmonary artery is ligated.

CONCLUSIONS

Ligation of the branch of the pulmonary artery to an affected lobe may be indicated in cases of chronic pulmonary suppuration to control recurring hemorrhage, if other methods have failed.

Phrenic neurectomy preliminary to, or at the time of, ligation will tend to decrease the development of collateral circulation from the bronchial artery with subsequent recurrence of pulmonary hemorrhage.

Failure to control pulmonary hemorrhage will follow ligation of the pulmonary artery if the bleeding is from a bronchial vessel.

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ABSCESS OF THE LUNG FOLLOWING FRACTURE OF THE RIBS

CONRAD GEORG, JR., M.D.
ANN ARBOR, MICH.

Abscess of the lung as a complication of fracture of the ribs is observed so seldom in medical and surgical practice that it seems proper to bring the report of such cases to the attention of this society. Careful search of the literature reveals the reports of only three cases. One of these was reported in 1896 by G. Jameson Johnston [1] of Richmond Hospital. In this case, the abscess followed fracture of the first rib. In another case, reported by Morton [2] for C. F. Pickering of Bristol, a man, aged 60, fell on his right side, injuring his right wrist. Chest symptoms did not develop for a week, then abscesses at the root of the neck and at the right sternoclavicular articulation occurred. The latter opened. Aspiration was performed on the chest, 26 ounces of pus being removed on one occasion and 20 ounces on another. The first right rib was fractured at the junction of the ossified cartilage with the sternum and 1 inch (2.5 cm.) farther out, beyond the chondrosternal junction. The third case was reported by Duff S. Allen,[3] of St. Louis, in a paper read before this society on May 9, 1927.

The etiologic factor in these cases was undoubtedly embolism. This infection is brought to the lungs through the blood stream. The most common cause of abscess of the lung, however, is aspiratory. According to Lilienthal,[4] the blood-borne infection begins in the parenchyma of the lung, and the aspiratory form begins in the bronchial tree and its ramifications. Infection may also take place by extension from neighboring tissues. The clinical studies of Richardson, Whittemore, Moore, Singer and Graham, Cutler and Schlueter, Hedblom and others show that a considerable percentage of all nontuberculous abscesses of the lung follow operative procedures on the upper respiratory tract. Cutler found that 29 per cent of 1,908 cases followed operations, half of which were tonsillectomies. Fetterolf and Fox expressed the belief that most abscesses of the lung following tonsillectomies are due to emboli dislodged from the paratonsillar tissues. These infectious emboli may lodge

1. Johnston, G. Jameson: A Case of Fracture of the First Rib, Complicated by Abscess Opening into the Lung, with Remarks on Fractures of the First Rib, Dublin J. M. Sc., Sept., 1896, vol. 102.

2. Morton: British M. J., Feb. 25, 1893.

3. Allen, D. S.: Etiology of Abscess of the Lung, Arch. Surg. **16**:191 (Jan.) 1928.

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in the radicals of the pulmonary arteries, causing infarcts which may soften later and form abscesses. Similar abscesses have been produced experimentally. Cutler and Holman, Weidlein and Schlueter produced them by using a segment of vein containing various kinds of bacteria. It has also been demonstrated by many surgeons that infection may take place by way of the lymphatics. Cutler and his associates have established the fact rather well that most abscesses of the lung after tonsillectomies result from septic emboli. Weil stated that embolism is usually the cause of chest symptoms which develop suddenly about ten days after an accident. Ochsner and Nesbit [5] said that a pulmonary abscess following tonsillectomy may be the result of the passage of an infected embolus from the vessels of the neck to the lung as shown in a few cases in which multiple pulmonary abscesses were demonstrated. Suppuration of the lung rarely occurs primarily, but is usually the result of the infection of tissues previously damaged by disease, injury or changes in the circulation. Some infections spread along the subpleural tissues to the lungs. Abscesses of the lung following operations under local anesthesia are undoubtedly due to infections along the lymphatics. Suppuration may extend from the pleura to the lungs after injuries to the wall of the chest.

The following case is reported on account of its rarity, and because the condition was wrongly diagnosed by the staff of a local hospital as a case of carcinoma of the stomach; this resulted in the cessation of compensation payments until the correct diagnosis was made.

REPORT OF A CASE

J. H. S., a man, aged 48, childless, on April 18, 1927, while working for the Washtenaw Gas Company fell from a ladder. The physician who was called by the company found a fracture of two ribs on the left side. The patient complained of a good deal of pain in the right side of the chest during the first six weeks, but attempted to return to work on June 1. At 3:30 p. m. of the same day, a severe pain started in the abdomen, and he was taken to a local hospital. The pain in the abdomen continued for a week, and then appeared in the lower portion of the right lung. Two x-ray pictures were taken, and a diagnosis of cancer of the stomach was made. Upon the announcement of this diagnosis, the insurance company discontinued the payment of the compensation because the injury was not regarded as the cause of the patient's condition.

The case was referred to me on June 22, 1927. Examination showed that the patient was acutely sick, suffering with severe pain in the lower part of the right side of the chest and in the abdomen, accompanied by dyspnea. There was marked tenderness on pressure over the lower three or four ribs of the right side, and the intercostal spaces were obliterated. Puncture with a needle in the eighth intercostal space in the posterior scapular line revealed pus. One and a half quarts of foul-smelling pus was aspirated. As a result of this procedure, the patient experienced

5. Ochsner and Nesbit: Pulmonary Abscess Following Tonsillectomy: Preliminary Report, Arch. Otolaryngol. 4:330 (Oct.) 1927.

great relief from pain and dyspnea. On June 24, he was again able to eat good meals, whereas formerly this was impossible on account of the pain. He was considerably emaciated and had a feeble pulse.

On June 25, the pulse rate was 80 and improved in quality. The patient complained of a little pain at the site of the needle puncture. Percussion showed dulness over the lower portion of the right lung. On July 5, resection of a portion of the eighth rib in the scapular line was done, and a large amount of serous fluid was removed. A cigaret drain was inserted. On July 7, there was a considerable discharge of pus, and tube drainage was inserted. The temperature was 103 F.; the pulse rate 90, and the respirations 32. There were severe pains in the right part of the upper portion of the abdomen. The patient coughed considerably with the expectoration of bloody pus. The diagnosis of abscess of the lung was made, but postural treatment could not be used on account of the severe illness of the patient, as he was not able to lie in one position for any great length of time. On July 16, the urine was passed involuntarily, and there was profuse sweating. The pulse rate was 140 and the respirations from 50 to 60. The patient died on July 18 at 8:15 p. m.

Postmortem examination on July 19 showed that the left lung was adherent to the ribs posteriorly at the point of fracture. The sharp edges of the fragments could be felt. Union had not taken place in the fracture as there was no formation of callus over it. The right lung had numerous adhesions posteriorly, where an abscess cavity was found. Both lungs floated in water, and pus escaped from the larger bronchi on pressure.

On July 23, Dr. A. S. Warthin, pathologist at the University of Michigan, who examined the tissues which were submitted to him for examination, reported that in the lungs there were multiple abscesses and gangrene. Purulent aspiration pneumonia was present. Colonies of mouth organisms were found in the bronchi. Diffuse congestion and edema and severe fibrinopurulent pleuritis were present. Passive congestion, slight cloudy swelling, fatty degenerative infiltration and early brown atrophy were observed in the liver.

Thus it appeared that the abscess of the lung was a complication of fracture of the ribs, and that the infection took place by embolism from the site of fracture. After the submission of this pathologic report, the insurance company granted full compensation to the widow.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. BRUNN, LAMBERT AND WEEKS, BEYE AND GEORG

DR. E. W. ARCHIBALD, Montreal: Dr. Brunn's paper interested me very much. While in San Francisco about two months ago I talked with him concerning his procedure. His series of six lobectomies with one death is, I believe, that which to date gives the lowest mortality figures. We have all been afraid of performing lobectomy. The problem of getting a low mortality rate in lobectomy has puzzled the members of the Society for years.

The series is still small, and one must suspend judgment as to the final value of the method. I understand, in fact, that a very recent patient died after Dr. Brunn left home.

What strikes me particularly is that, whereas Dr. Robinson, Dr. Graham, Dr. Whittemore and myself insist on the general principle of creating adhesions first, in order to prevent empyema, and whereas Dr. Whittemore and I, in that endeavor, believe in the value of exteriorization, partial or complete, of the lobe to be resected, Dr. Lilienthal and Dr. Brunn following him have gone to the

opposite extreme; that of a one-stage operation through the open pleura, taking a chance on tension pneumothorax and on infection, whether of the pleura or mediastinum. The danger of mediastinitis is possibly equally great with both methods, but of the pleura it is certainly not. Dr. Brunn expects the late development of empyema, but he does not fear it.

I confess I have been much struck with the results which Dr. Brunn has obtained. I must revise my ideas considerably about the danger of empyema under these circumstances. Dr. Brunn aims at localizing the empyema by keeping up a constant aspiration of the collecting pleural fluid and air, and in this sense allowing the upper lobe to come out and become fastened to the upper part of the pleura. He overcomes tension pneumothorax in the same way by depending on the bronchial ligature to keep air out of the pleural cavity for from five to seven days after operation in which time the upper lobe is adherent and serous pneumothorax impossible.

The only faults that I can see in the argument is that the adhesion of the upper lobe will not occur in the five to seven day period, and when the bronchial stump ligatures are absorbed or cut through an acute tension pneumothorax, and probably an acute widespread empyema, will occur possibly involving the whole thorax. That danger, I still fear. Nevertheless, it is impossible not to admire the courage with which Dr. Lilienthal and Dr. Brunn tackle this danger and the skill with which they remove a lobe, close the thorax and get satisfactory results. But I must also admire the patience of Graham with his cautery method and one must remember that counting this last death of Dr. Brunn's list (two deaths in seven cases), Dr. Graham's figures are still a little better. I was very much struck with the principle of treatment adopted by Dr. Brunn with regard to bronchial fistula. The continuing open bronchial fistula has been a bugbear to all of us. Various operations have been devised to close them, Dr. Brunn's way is a simple one—leave them deep, and they close themselves. The uniform closure of bronchial fistula in his reported cases is sufficient evidence of the truth of this proposition.

All in all I confess that I am thrown back a bit. I did hope that by exteriorization we had evolved a principle which would give a very low mortality rate. Dr. Whittemore can speak concerning that subject with more experience than myself. For the next few years two opposing methods will compete for favour; that of excluding infection by exteriorization of the lobe and that of trusting the pleura and doing the one-stage operation and closing the thorax.

I feel that Dr. Brunn's work in his series represents a step forward, and I hope that by next year he will be able to report to us a larger series with a still better mortality rate.

Dr. Howard Lilienthal, New York: I think that Dr. Brunn's results were satisfactory. Those who died could not have recovered and were in a horrible condition anyway. Those patients who recovered are well and not merely relieved.

I would like to call attention to one or two points. In the first place, I feel greatly encouraged by the increasing interest in lobectomy in properly selected cases. I will not here enter into the question of proper selection, but you all probably know what I mean—I am very much pleased to think that in the cases presented by Dr. Brunn lobectomy has been chosen as the correct procedure and that it is being more frequently selected than has been the case in the past few years. In New York, Dr. Neuhof and others have been doing similar work.

Dr. Brunn has spoken about nasal sinuses. It is most important that the nose should be examined before the operation, and that the source of infection should first be done away with if possible.

Concerning air leakage, little chance is taken by the method that Dr. Brunn has devised, which is by a tube drainage with suction. I believe that Dr. Brunn, after I have talked with him a little while, may substitute his syringe suction by a rubber valve suction made with a finger cot. There the slightest respiratory motion will permit the egress of air from the chest and will also prevent air from entering. It is better than a water-seal drain. Then when the time has come for the stump to atrophy or slough and the bronchi to open, there is no danger from acute tense pneumothorax. I think one important contribution that Dr. Brunn has made is in the avoidance of the use of gauze packings. I am sure that the use of gauze, even a fillet down to the stump, in cases in which I have performed an operation, has contributed to failure. His principles are excellent.

In regard to fracture of the ribs, I would like to ask whether there was any osteomyelitis of those ribs?

DR. CONRAD GEORG: There was not.

DR. HOWARD LILIENTHAL: Then may I suggest that the abscess of the lung resulted from the same accident which produced the fracture of the ribs but probably did not come from the fracture itself.

DR. EVARTS A. GRAHAM, St. Louis: I have been interested in Dr. Archibald's remarks and discussion. I think, perhaps, that I might differ from him in his final conclusions. It seems to me that the type of case in which he suggested that lobectomy rather than some other method will perhaps be used more frequently in the future is the kind of case which has placed lobectomy in ill repute in the past. This type of case is accompanied by small abscesses and, as Dr. Brunn brought out, has almost universally been fatal following lobectomy. It has been in this type of case, so far as I know, that the only satisfactory method of treatment has been some such method as cautery pneumectomy. I do not wish to advocate that procedure to the exclusion of other methods. I have always looked on it as imperfect. I think in nearly every article in which I have mentioned that method I have stated that I regarded it as not the final development in the treatment for these conditions, but only as a step toward a better solution of the problem. I do feel, however, that it would be unfortunate for humanity to go back and to begin its experience all over again in regard to this type of case. I think that so far as the use of lobectomy in conditions of multiple abscess is concerned, it would be most unfortunate. I believe I am correct in my recollection of Dr. Lilienthal's series that it was that type of case which was preeminently fatal, and I am sure that I recall Dr. Hedblom's statement that in his series of thoracoplasties it was almost exclusively that type of case which was fatal. The principal difficulty with lobectomy is its high mortality rate. A few successful results should not blind one to the fact that in general the operative mortality has been very high. It does not seem to me that Dr. Brunn's results in a few cases warrant any great optimism about the safety of that operation.

DR. LEO ELOESSER, San Francisco: Just a few words, perhaps, of conciliation. I think that in theory we all agree that in the type of case under discussion lobectomy is the ideal to be striven for, and that we are probably passing through a stage in operative procedures on the chest such as has been passed through in abdominal surgery. I think probably that the reason for the dispatch with which Dr. Brunn has been able to conclude his operations and which probably makes his operative mortality so low is the fact that he operates without the aid of pressure apparatus in a pneumothorax with a collapsed lung; for access to the lung is much better with a collapsed lung than with a distended one. How far we are going to be able to equal the brilliancy of his results remains to be seen, but it seems to me that in theory probably it is a goal to be striven for.

I wish that Dr. Graham had told us something about the end-results in the cases in which he has performed pneumectomy, because the few patients whom I have followed have in the end become impatient of the endless cauterizations; furthermore, I was unwilling to let them return home with open fistulas. I would like to hear the end-results in cases in which Dr. Graham has the lobe wide open. It seems to me, also, that there is a middle course which one can take, one between Dr. Whittemore's procedure and that of Dr. Brunn, even for patients who have had previous suppuration and whose lobes are carnefied and show the shadows that have been the bane of Dr. Hedblom and of everybody else. This procedure is something like the one Dr. Whittemore has advocated, which really amounts to a two-stage lobectomy. This operation is possible if one will clamp off the pedicle, and then either immediately at the first stage or subsequently remove the lobe, packing the pedicle about with gauze and allowing the wound to remain open, using the skin of the wall of the chest to make a compartment, as it were, to separate the sound part of the chest from that which has been sealed off by previous adhesions.

DR. EVARTS A. GRAHAM: I have been asked to say something about the results of cautery pneumectomy. These results have been published as recently as last January, in the *American Review of Tuberculosis.*

I would like to say, in addition, that I think that a two-stage lobectomy, when it can be done safely, is always an excellent procedure. Indeed, I think that a one-stage procedure is excellent if it can be done safely. The most essential requirement necessary to establish lobectomy as a satisfactory procedure is that it can be done with reasonable safety. I think that with all of these methods it will be necessary to wait a period of years until a sufficient number of cases are collected in order to know which is the most effective. Certainly a simple type of operative procedure cannot be satisfactorily applied to all types of chronic pulmonary suppuration.

Physicians write or talk to me once in a while about the cautery operation, and conversation is often somewhat as follows: "I have tried your operation in three cases, in one case the results were excellent, in another a fistula occurred, and in the third case the patient died." "Well, what did he die of?" "Oh, he just died." The conversation generally ends with: "I therefore regard the operation as an unsatisfactory procedure." Well, I would regard the use of that operation in cases which are evidently like the ones that Dr. Brunn has been talking about as perhaps not altogether successful. In my own experience, the cautery operation has been applied almost solely to the worst type of pulmonary suppuration with which one has to deal, and in three instances of bilateral chronic suppurative pneumonia it has been applied with successful results. In dealing with cases of this kind, I do not see how one can expect to get results which at all approach those obtained in operations in other regions of the body. My own experience has been also that practically all patients who are not treated will eventually die from the effects of pulmonary suppuration. They die of infection, as Dr. Archibald mentions; they eventually die of cerebral embolism, and they die also of hemorrhage. I am, of course, excluding from consideration those patients who have acute abscesses of the lung, many of whom recover spontaneously. Using the hypothetical instance I mentioned, I am not sure that the method should be condemned because in this individual's experience one patient of three died after the performance of the operation. I am not sure that the patient died as a result of the operation.

In my own experience during a period of nearly four years, 24 per cent of forty-five patients on whom I have used this method have died; but only 6.6 per

cent died in the hospital. The others died from various causes after leaving the hospital; in some cases I am not sure what the causes of death were, as in some instances it occurred two and three years after the patients were discharged. I therefore do not feel that the mortality rate in my cases is particularly high for a type of surgical case which to my knowledge is the most desperate, except cancer, so far as spontaneous mortality is concerned.

I do not wish any one to understand that I am taking the position of an advocate for this particular operation, as I am not. I am glad to use other methods. As I said, I have applied this method only to what I regard as cases of the most desperate type of pulmonary suppuration, in which I think a good many members of this Association would refuse to perform an operation otherwise, and therefore I am not particularly disappointed in the results that have been obtained.

DR. HAROLD BRUNN: I feel that the few cases which I have reported, and in which operations were performed over a rather long period of years, with a gradual change in technic, give one certitude as to the operative mortality. Nevertheless, I believe that certain points in the technic are worthy of consideration, and I trust that further improvements will so lower the mortality that thoracic surgeons may use this operation more frequently than they have in the past.

In the discussion as to the type of case which I consider favorable for lobectomy, I have already stated in my paper that the cases reported were true cases of bronchiectasis and not cases of the honey-comb abscess type. They were cases in which there was a long chronic history and in which there appeared to be no prospect of permanent cure. As to time of operation, I chose to operate not during any acute exacerbation, but rather after the patient was in as good physical condition as seemed possible.

On inquiry, I find that surgeons in clinics where surgical measures are performed on the lungs seldom use lobectomy; perhaps Dr. Lilienthal, of New York, may be excepted. It seems to me that every one performing operations on the chest must at certain times in an exploration come to the conclusion that lobectomy is the operation of choice, and I feel that in the next few years the mortality will be lowered sufficiently so that when the occasion arises this procedure will be chosen with every assurance of success. I admit the usefulness of cautery pneumonectomy, multiple stage thoracoplasty and the exterioration operations, but I feel that lobectomy should not be eliminated from the field of lung surgery as it has been in the past.

UNILATERAL PNEUMOTHORAX

THE BEHAVIOR OF THE MEDIASTINUM

HOWARD LILIENTHAL, M.D.
NEW YORK
AND
J. BURNS AMBERSON, JR., M.D.
LOOMIS, N. Y.

When the thorax has been opened for the drainage of an acute empyema, almost invariably there is a gush of pus under tension. During inspiration, there is a sound of suction as the air rushes into the chest; during expiration, straining or coughing, the pus and air are forcibly expelled through the thoracotomy wound. This is partly explained by the fact that during inspiration the entire thorax increases in capacity, while during expiration its volume decreases. There is, however, another cause for the violent ejection of fluid and air, and this can be easily demonstrated by the fluoroscope, or, more strikingly, when a wide thoracotomy with free exposure of the lung has been made. It will be seen in both instances that the lung of the open side increases in volume with forced expiration, while it decreases on inspiration. If the mediastinum is mobile, it will be deviated toward the open side on expiration and will flap toward the normal side on inspiration.

In determining the prognosis in cases of thoracic fistula following empyema, fluoroscopic observations made during inspiration and forced expiration will demonstrate this motion of the lung, and, if it is not rigid, of the mediastinum as well. The greater the motion, the better is the chance of curing the patient of the empyema.

When the chest is normally closed, however, and for some reason the lung of one side is collapsed by air within the pleural sac not connected with a bronchus, the motions of lung and mediastinum on expiration are exactly opposite to those which occur when the chest is open. On inspiration and on straining with the closed glottis, the mediastinum does not move.

Few had given thought to this, and it was unexpectedly observed when the deviation phenomenon was demonstrated in a case from the service of Dr. B. S. Oppenheimer in the wards of Mount Sinai Hospital during the past winter. Observed by the roentgenologic staff of the hospital, the mechanics of the mediastinal deviation on deep inspiration and forced expiration became the subject of discussion. Indeed, this case (figs. 1 and 2) was demonstrated by Dr. Emanuel W. Benjamin at a clinical conference of the hospital, when its apparently unusual character excited much general interest. Fluoroscopic examination was then made in other cases of closed pneumothorax, and it was found that the

mediastinum moved with respiration. In deep inspiration, it usually seemed to remain in the midline, but deviated strongly toward the normal side on full expiration. On straining with the glottis closed, the organs within the mediastinal space were not displaced.

Considering this as a problem in physics, it at once became evident that the reason for the deviation was that while the air within the normal side could be expelled through the glottis, the imprisoned air of the pneumothorax could be reduced in volume only slightly by compression when the walls of the chest approached each other, and, quite naturally, this body of air under pressure would force the normal mediastinum and its organs toward the well side, at the same time driving the air out of the normal lung by further reducing, from the mesial aspect, the size of the already contracted normal hemithorax.

From the wealth of material at the Loomis Sanatorium, a number of cases of artificial pneumothorax were selected for study, and a number of interesting observations were made. This work was done at Dr. Amberson's request by Dr. George Foster Herben, associate physician to Loomis Sanatorium.

As the cases from Loomis were artificially produced, they are almost ideal for observations of this kind, a single weak point being the presence of a diseased lung on the side of the pneumothorax; but this would tend rather to stiffen than to mobilize the mediastinum, so that any possible error would not invalidate the conclusions.

Dr. Herben came independently to the same conclusion that we had reached concerning the mode of production of mediastinal deviation, and he has also noted other interesting facts included in the following paragraphs, which are quoted verbatim:

> In the course of routine fluoroscopy of the patients undergoing therapeutic pneumothorax at Loomis Sanatorium, the phenomenon of so-called "shifting mediastinum" was frequently encountered. Shadows on the "good" side were noted during full expiration in a number of instances in which dissimilar shadows appeared during deep inspiration. The present series of films was made to ascertain the degree of mediastinal shift and to determine, if possible, the mechanism producing this prenomenon.
>
> Of some 34 cases studied substantial mediastinal shift was noted in 15. Of these 15 patients, 4 had right-sided pneumothorax and 11 had left-sided pneumothorax.
>
> The degree of mediastinal displacement was estimated relatively in each case by noting the distance of the point "farthest down and farthest out" of the cardiac shadow from the midline of a film exposed during deep inspiration and comparing this distance with that similarly obtained of a film made immediately thereafter during full expiration. The mechanism producing the shift is based on the presence of a fairly complete pneumothorax, uninfluenced by pleural exudate, massive pleuropulmonary or pleurodiaphragmatic adhesions or a combination of these elements. A nonrigid mediastinum is presupposed.
>
> 1. Deep inspiration being produced by maximum descent of both the leaves of the diaphragm and coincident enlargement of the anteroposterior and lateral

diameters of the bony thorax the "good" lung passively fills as completely as possible and a minimum pressure, less than atmospheric, exists in the contralateral pneumothorax space. In full inspiration the mediastinum is observed to occupy what is believed to be its normal position.

2. Forced expiration increased the pressure in the pneumothorax. The air contained therein has no outlet and, as the thoracic dimensions diminish, the pressure in the pneumothorax progressively increases. The pressure exerted upon the "good" lung, however, diminishes its bulk by driving the contained air out through the open glottis. The increasing pressures in the closed pneumothorax are exerted partly upon the mediastinal structures, which if not well fixed must shift toward the good side, thus further decreasing the bulk of the good lung. Obviously, if some air is contained in the compressed or bad lung a portion of this air also will be expelled. The amount of air contained in the compressed lung, however, is always relatively much less than that contained in the good lung, and the increasing pressures of the air contained in the pneumothorax space serve to compress and displace the compressed lung toward the "good" side as the mediastinum shifts in advance of the compressed lung.

With a mobile mediastinal structure, the compressed lung in its motion toward the good side in expiration will be forced to occupy what space it may. In those cases in which a flexible mediastinum exists, it has been observed that the retrosternal space, i. e., that portion of the chest between the anterior surface of the fibrous pericardium and the posterior surface of the sternum and costal cartilages, is invariably occupied by the compressed lung to an increasing degree as the pressures deviating it toward the good side progressively increase. In some instances in this series, the degree to which the left lung usurped the retrosternal space, and even occupied a portion of the opposite or good hemithorax is remarkable.

There was noted a definite difference in the behaviour of the shifting mediastinal structures when right-sided pneumothorax cases were compared with the left-sided ones. With right-sided pneumothorax the mediastinal structures are displaced considerably into the left side of the chest, and since the heart is already well to the left of the midline, increasing pressures from the right cause further displacement to the left with rotation of the heart backward and inward along the lateral surface of the spinal column. That this rotation occurs is strongly suggested by the decrease in the transverse diameter of the heart shadow when the film on full expiration is compared with that on inspiration. This phenomenon is less striking when pneumothorax pressures in the left-sided cases give a shadow of the mediastinum to the right. Here the tendency is to make the heart assume a midline position, wedged between the vertebral column and the sternum. In only exceptional instances has it been observed that the shadow of the heart to the right appears as striking as does the shadow to the left. Rotation of the heart is definitely less marked in cases of left-sided pneumothorax than in right-sided ones.

In the *Revue de la tuberculose* for October, 1926, there is an article by Edgard Eber in which will be found a bibliography with eighteen references. The reader is also referred to Ludwig von Muralt's work, Der Kunstliche Pneumothorax, second edition by Karl Ernst Ranke, Julius Springer, Berlin, 1922. Other authors have also written on the subject from time to time. We merely wish to present here some independent observations and conclusions which it seems worth while to place on record. We would especially accentuate the difference in respiratory mechanics in open and closed pneumothorax. We have not taken up the

question of pleuropulmonary fistula, nor have we gone into the finer points involving the elasticity of lung tissue. In any event, pneumothorax clinically studied, presupposes an abnormal condition in some portion of the lung on the affected side.

For the purposes of this paper the diaphragm, except in figure 5, *A* and *B*, is considered as acting normally on both sides. In this figure, the slight difference due to a unilateral partial paralysis does not materially affect our conclusions. There has been no case of high tension pneumothorax which of itself might dislocate the mediastinum. All x-ray exposures were made with the patient in an upright position.

ILLUSTRATIVE CASES

The following illustrations depict the behavior of the mediastinum when unilateral pneumothorax has been induced.

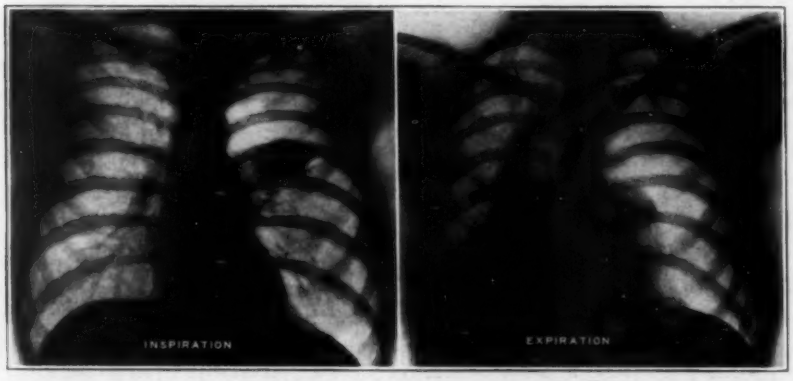

Fig. 1.—Spontaneous pneumothorax. In the figure on the left, taken during deep inspiration, both lungs are expanded, that on the abnormal side not fully. The mediastinum is little or not at all deviated to the abnormal side. In the figure on the right, taken from the same case during full expiration, the mediastinum is strongly deviated to the normal side, compressing the lung to a "pneumonic shadow" in the lower portion.

In the following illustrations, as in this, the figure on the left is taken during inspiration and that on the right, during expiration.

CASE 6.—*Inspiration and expiration noted postero-anteriorly.*

Left-sided pneumothorax was induced in a man, aged 26, for the control of a slowly progressive cavitary lesion which failed to show adequate repair after the patient had spent a prolonged period in a sanatorium. The duration of the pneumothorax was eight months. From 300 to 600 cc. of air was given, usually 500 cc. The maximum end-pressure was —2. For the past two months before the case was reported tests of the sputum were negative. The roentgenograms showed a shift of the mediastinum 3.5 cm. to the right with elevations of the left side of the diaphragm the distance of one rib and one interspace. As in case 4, considerable shifting occurred, despite the fact that positive pressure in the left pleural space had never been employed. As in the other cases in the series, the swing of the tracheal bifurcation was well shown.

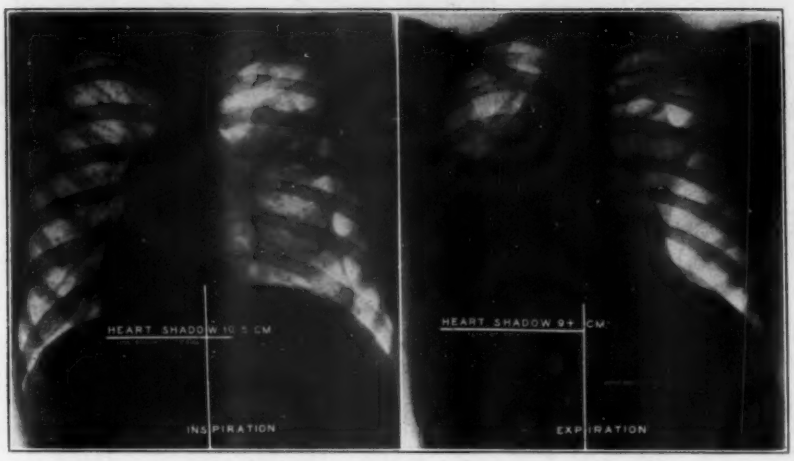

Fig. 2 (case 1).—Inspiration and expiration, postero-anterior view of a man, aged 27. Right-sided pneumothorax induced for progressive unilateral disease with increasing cavity formation. Duration of pneumothorax, six months. Amounts of air given from 300 to 600 cc., usually about 400 cc. Maximum end-pressures given, + 3 cm. of water. Tests of sputum still positive. The roentgenograms show a deviation of the mediastinum to the left, just 3 cm., rotation of the mediastinum posteriorly into the left side of the chest being shown by diminution of the heart's greatest transverse diameter from 10.5 to 9.0 cm.

Fig. 3 (case 2).—Inspiration and expiration, postero-anterior view of a man, aged 29. Left-sided pneumothorax induced for progressive unilateral disease with increasing cavity formation. Duration of collapse, twelve months. Amounts of air given, from 150 to 600 cc., usually about 450 cc. Maximum end-pressures, + 3 cm. of water. Tests of sputum still positive. The roentgenograms show a deviation of the mediastinum to the right of 4.5 cm. without noteworthy diminution in the greatest transverse diameter of the heart. This maintenance of the transverse diameter practically unchanged is the rule with mediastinal displacement from left to right. Note especially the striking shift of the tracheal bifurcation and the marked increase in the apparent size of the left pneumothorax space.

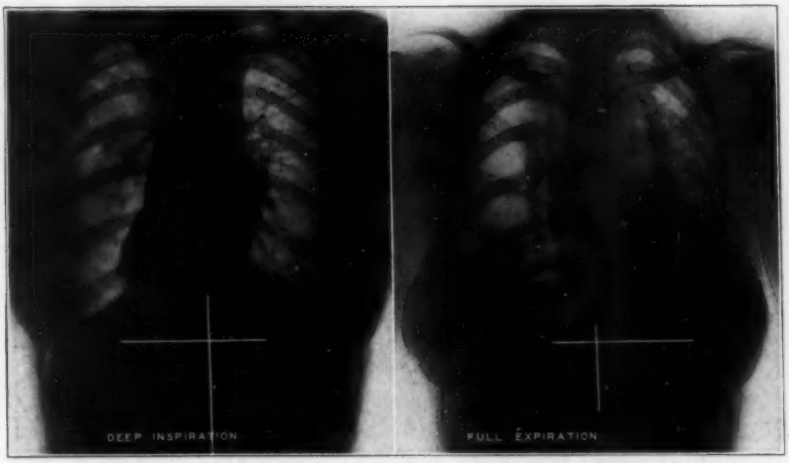

Fig. 4 (case 3).—Inspiration and expiration, postero-anterior view of a woman, aged 21. Left-sided pneumothorax induced for progressive unilateral disease with increasing small multilocular cavity formation. Duration of collapse, fifteen months. Amounts of air given from 200 to 400 cc., usually from 250 to 300 cc. Maximum end-pressures given, + 4. Sputum gave negative reactions for five months. The roentgenograms show a deviation of the mediastinum to the right of 4 cm. with a slight diminution, 0.5 cm. in the greatest transverse diameter of the heart. Here is most strikingly shown the marked shift in the tracheal bifurcation, which is even greater than that observed in case 2. Note especially the decreased penetration of the rays through the base of the right lung, the appearance of which on forced expiration suggests that seen in atelectasis.

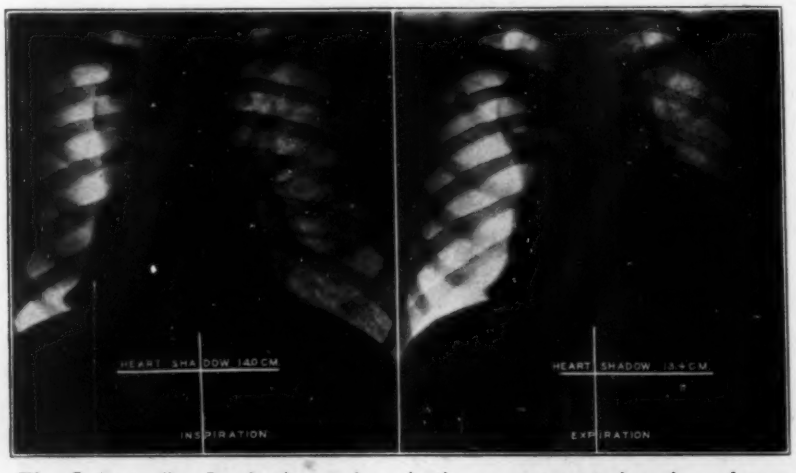

Fig. 5 (case 4).—Inspiration and expiration, postero-anterior view of a man, aged 24. Left-sided pneumothorax induced for progressive unilateral disease with increasing cavity formation. Swaying mediastinum present from start of pneumothorax. Apical cavity held open by adhesions. Left phrenicotomy performed following three months' pneumothorax. Duration of collapse, nine months. Amounts of air given, from 400 to 500 cc., usually about 400 cc. Maximum end-pressures, — 1. Tests of sputum remained positive. The roentgenograms show a shift of 4 cm. to the right, the apical cavity still patent, due to adhesions; the left side of the (paralyzed) diaphragm is at same level as the right (top of sixth costal cartilage) on inspiration. On forced expiration, the paralyzed diaphragm was elevated about 1.5 cm.; the right side of the diaphragm has been elevated to the level of the upper border of the fifth costal cartilage, a distance of nearly 4 cm. Paralyzing the left side of the diaphragm has not sensibly affected the compression of the left lung, which, due to the swaying mediastinum, still possesses considerable mobility. The presence of a nonrigid mediastinum largely offsets whatever advantages the phrenicotomy might otherwise afford. Note that in this case positive pneumothorax pressures have never been given.

Fig. 6 (case 5).—Inspiration and expiration, postero-anterior view of a man, aged 27. Right-sided pneumothorax induced for hemoptyses, otherwise uncontrollable, and to diminish danger of bronchogenic spread. Duration of pneumothorax, twelve months. Amounts of air given, from 150 to 550 cc., usually about 350 cc. Maximum end-pressures, + 6, usually + 2. Sputum negative for bacilli for six months. The roentgenograms show a deviation of the mediastinum into the left side of the chest of 2.5 cm. and a diminution in the greatest transverse diameter of the heart from 12 cm. to about 9 cm. This case shows more strikingly than case 1 the more pronounced effect on the heart and the degree of torsion produced by the encroachment of the right lung on the heart when acted on by increased pressure in closed right pneumothorax space.

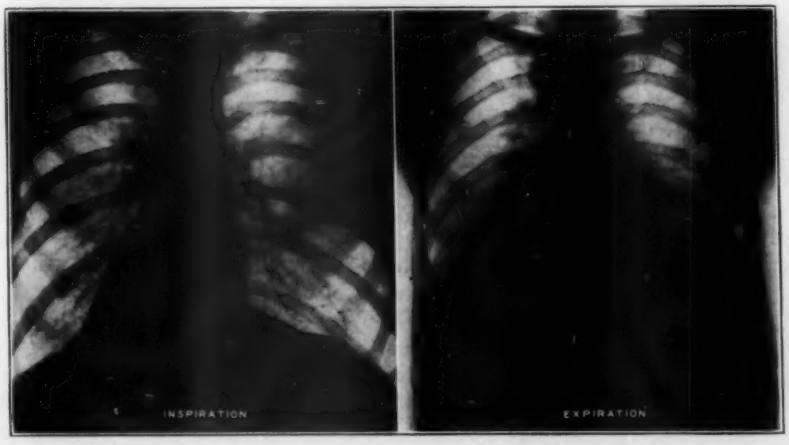

Fig. 7 (case 7).—Inspiration and expiration, postero-anterior view. Roentgenograms of a presumably healthy chest taken under precisely the same conditions as the other roentgenograms of the series. No mediastinal shift is revealed, but the marked alteration in the contour and diameter of the wall of the chest and of the heart shadow are shown. Note especially the diminished illumination at the bases during expiration, suggesting the changes seen in partial atelectasis.

CONCLUSIONS

1. In pneumorthorax with an external opening, blowing exercise tend to inflate the collapsed lung and to force the mediastinum toward the open side.

2. In closed pneumothorax, blowing exercises tend to compress the collapsed lung, and the mediastinum is forced toward the healthy lung, which is also compressed.

3. In pneumothorax with an external opening, straining with the closed glottis deviates the mediastinum to the open side and expands the collapsed lung.

4. In closed pneumothorax, straining with the closed glottis produces little or no deviation of the mediastinum.

5. In closed pneumothorax, forced expiration tends to rotate the heart and its attachments.

6. In roentgenologic study of pneumothorax, closed or open, observations and records should be made in deep inspiration, in full expiration and in straining against the closed glottis.

ABSTRACT OF DISCUSSION

DR. AMBERSON, JR.: My interest is chiefly in mediastinal movements in artificial pneumothorax as used in the treatment of patients who have pulmonary tuberculosis. Shifting of considerable degree is encountered most frequently in those cases in which the disease has not caused sufficient thickening of the mediastinal pleura to bring about its fixation. In some cases the shifting is so wide as to cause an encroachment on the respiratory capacity of the opposite functioning lung, in which case frequent fluoroscopy is necessary to careful adjustment of the intrapleural pressure. Otherwise, mere shifting of the mediastinum does not appear to be dangerous, since other accidents, such as mediastinal rupture, are extremely uncommon.

One of the chief disadvantages of a swaying mediastinum is that it may interfere with securing a collapse of the tuberculous lung sufficient to produce the desired therapeutic effect. As Dr. Alexander has pointed out, mere physiologic rest suffices in some cases to bring about the desired results; but as a rule, effective collapse of tuberculous cavities is necessary. In about 300 cases at the Loomis Sanatorium, which were followed for from five to fourteen years, we found that almost three times as many patients in whom a complete collapse of the lung was obtained were alive as patients in whom the collapse was insufficient. It is essential, then, in order to get the best results, to strive for the obliteration of cavities. As has been shown in several of the cases illustrated here, a swaying mediastinum prevented this. Greater success would most likely be obtained in such cases if the mediastinum were stiff enough to permit the inducing of positive intrapleural pressures by the injection of air. Such pressure brought to bear on cavity walls often effects the obliteration, which otherwise is unattainable. When the mediastinum sways, such positive pressures reduce the respiratory capacity of the functioning lung; consequently, one compromises and the results are not the best.

In some of these cases we welcome the development of a pleural effusion, provided it is of the usual mild type, and at times, while we have never done it,

we have been tempted to inject some sort of an irritant to induce an effusion. The reason for this is that following such a serofibrinous pleurisy, inflammatory thickening may stiffen the mediastinum, thus permitting the use of positive intrapleural pressures.

DR. LILIENTHAL: As I said before, I was astonished when I found that what ought to have been expected really did happen in closed pneumothorax. I had become so accustomed to working with open pneumothorax that when I first saw the x-ray pictures of a closed one, I was much excited about it. I have found that others have written on the subject, but at the same time I do not recollect any mention of the rotation of the heart or its attachments, nor have I seen pictures such as those which have been displayed here.

EFFECTS OF A LARGE CLOSED BILATERAL PNEUMOTHORAX ON THORACIC LYMPH FLOW *

FRANK S. DOLLEY, M.D.
LOS ANGELES
AND
E. ROBERT WIESE, M.D.
INGOMAR, PA.

Our experiments lead us to believe that an acute closed bilateral pneumothorax of considerable amount as induced in dogs causes not only a marked decrease in lymph flow from the thoracic cavity, but also greatly retards absorption of colloid material from the pleural spaces. If this be true in man, we deem it of prime importance to consider this fact in the treatment of intrathoracic disease.

METHODS

Our first concern was to assure ourselves that we were actually measuring approximately all the lymph issuing from the left thorax in the dogs on which we were working. This was by no means a simple matter. There were several complicating factors. In the first place, the locations, courses and anastomoses of the thoracic duct of the bronchomediastinal, jugular and subclavian lymph vessels are notoriously variable (fig. 1). Sometimes all these vessels on the left side united to form an ampulla continuous with the vein itself. Occasionally, all the lymph vessels joined the veins separately; any two or three might unite before final venous communication. The distributions were most bizarre, yet it was found that no matter how aberrantly they might conduct themselves where contacts were many, they could be depended on to discharge their lymph, by one trunk or many, as the case might be, at the venous angle caused by the confluence of the left internal jugular and left subclavian veins—the angulus venosus of Ghon.[1]

Again, no one lymph-collecting trunk carries away the lymph from the right side of the chest or from the left side. A considerable amount of lymph drainage from the pleural walls occurs by way of the intercostals. The lymph from these reaches the left venous angle by three routes: (1) Anteriorly, the intercostals drain into the internal mammaries and from thence into the bronchomediastinal vessels; (2)

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* From the Department of Surgery, Washington University School of Medicine and Chest Service of Barnes Hospital.

1. Ghon, A.: Die Veränderung der Lymphknoten in der Venenwinkeln bei Tuberkulose und ihre Bedeutung, Ztschr. f. Tuberk. **46**:97, 1927.

laterally and posteriorly, the internal intercostals drain into the thoracic duct, and (3) the external intercostals empty via the axillary nodes into the subclavian lymph vessels. Absorption of lymph from the diaphragm, visceral pleura and lungs takes place through the bronchomediastinal vessels and the thoracic duct. Thus these two and the subclavian vessels serve to a greater or lesser degree as evacuation canals for the left side of the chest, and discharge at the left venous angle.

Finally, a complicating factor is the amazingly rich lymphatic anastomoses. Valves, though present in the main collecting trunks and though numerous in the various smaller vessels and in certain diverting locations in plexuses, as a rule, prevent retrograde, but not lateral, flow, so that any obstruction in lymph advance is cared for easily and quickly

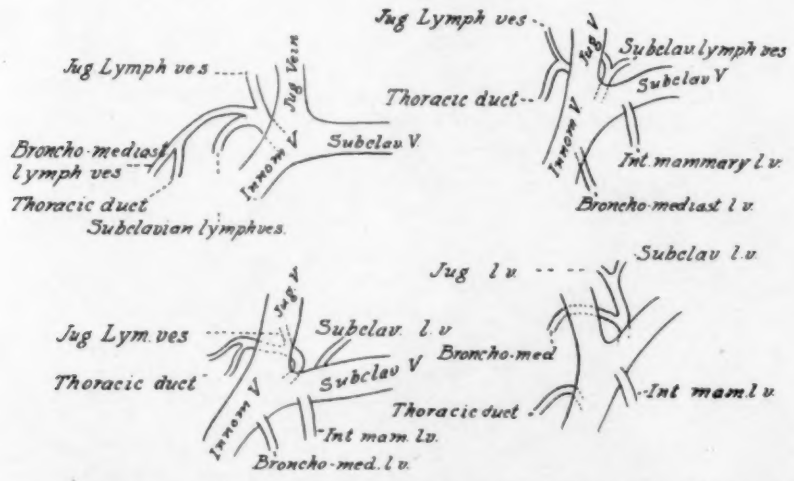

Fig. 1.—Great variability in course and insertions of lymph vessels at angulus venosus.

by collateralization and by passing into other trunks—an adjustment which under normal conditions safeguards tissue pressure.

Since approximately all the tributaries draining the left thorax empty close to the venous angle, a lymphatic cistern was created at this point, ligating first the left internal jugular and left subclavian veins and then the left innominate vein, thus excluding all blood but including all lymph draining from the left thorax. Cannulation of this pouch did not interfere with lymph flow; no back pressure into the lymphatic vessels occurred (fig. 2).

We employed the Singer pneumothorax apparatus in order to assure ourselves that we were in the pleural space. For colorimetric work, we checked against a color differential in which we used graduated known dilutions of the dye whose presence we were seeking to detect

in the lymph itself. In our earlier experiments the dogs were given one half pint of cream from one to three hours before the experiments began, in order to distinguish the thoracic duct more readily by the increased lacteal flow of milky lymph. In later work, after our technic was well established, not only cream, but the morning meal was withheld.

PROTOCOLS OF EXPERIMENTS

Experiments A.—Control work in which trypan blue was injected into the left pleural cavity but no pneumothorax instituted.

All control experiments were uniform and constant. Only one control experiment that is typical of all will be recorded here.

Dog 1.—Nov. 17, 1927: 8:30 a. m.: One half pint of cream given.

10:45 a. m.: Experiment began.

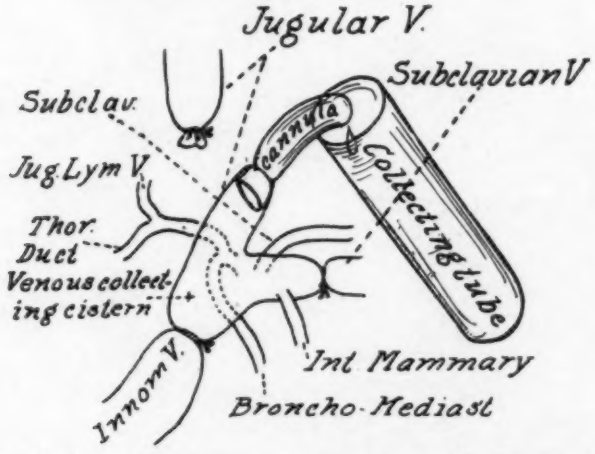

Fig. 2.—Method of formation of venous cistern for lymph collection.

11:10 a. m.: Flow of lymph from cannula in venous lymphatic cistern began; milky white.

11:42 a. m.: Two cubic centimeters of 1 per cent trypan blue injected into left pleural cavity.

12:04 p. m.: Blue color first discernible in lymph at cistern by colorimeter.

Twenty-two minutes after the injection of trypan blue in the left pleural cavity, blue appeared at the collecting cistern.

3:30 p. m.: Animal killed. There was little variance in flow and no appreciable change in amount of trypan blue in lymph during this time.

The autopsies on the experimental animals will be discussed as a whole when the report of experiments themselves is concluded.

Experiments B.—Measurement of rate of lymph flow into cistern, then trypan blue injected into left pleural cavity together with 250 cc. of air.

Dog 3.—Dec. 28, 1927: Animal given neither cream nor morning meal.

11:15 a. m.: Flow of lymph from cannula in venous lymphatic cistern began; clear amber.

11:45 a. m.: Total amount of lymph collected, 10.2 cc.

11:49 a. m.: Two cubic centimeters of 1 per cent trypan blue injected into left pleural cavity.

11:50 a. m.: Two hundred and fifty cubic centimeters of air injected into left pleural cavity. Respiration at once noticeably slowed and deepened. Type changed from thoracic to abdominal in character; no dyspnea. Lymph checked against colorimeter every five minutes for the detection of blue during experiments.

12:49 p. m.: Animal killed one hour after injection of trypan blue into pleural cavity and fifty-seven minutes after institution of 250 cc. pneumothorax. No blue detectable by colorimeter during this time.

Total amount of lymph flow into cistern for thirty-two minutes before pneumothorax, 10.2 cc.

Total amount of lymph flow into cistern for fifty-seven minutes after pneumothorax, 8.2 cc.

Dog 4.—Dec. 29, 1927: Animal given neither cream nor morning meal.

10:15 a. m.: Flow of lymph from cannula in venous lymphatic cistern began; clear amber.

10:45 a. m.: Total amount of lymph collected, 9.2 cc.

10:47 a. m.: Two cubic centimeters of 1 per cent trypan blue and 250 cc. of air injected into left pleural cavity. Breathing at once deepened and slowed.

11:35 a. m.: Slightest possible detectable trace of blue verified with colorimeter,

12 Noon: Animal killed, one hour and thirteen minutes after injection of trypan blue and air into the left pleural cavity.

Forty-nine minutes after injection of trypan blue and air into the left pleural cavity, blue appeared at the collecting cistern.

Total amount of lymph flow into cistern for thirty minutes before pneumothorax, 9.2 cc.

Total amount of lymph flow into cistern for one hour, sixteen minutes after penumothorax, 8.8 cc.

Dog 5.—Dec. 30, 1927: Animal given neither cream nor morning meal.

11:35 a. m.: Flow of lymph from cannula in venous lymphatic cistern began; clear yellowish amber.

12:05 p. m.: Total amount of lymph collected, 12.6 cc.

12:06 p. m.: Two cubic centimeters of 1 per cent solution trypan blue and 250 cc. of air injected into left pleural cavity. Breathing at once deepened and slowed.

12:50 p. m.: Blue color first appeared in lymph, verified by colorimeter; slightest possible trace.

1:30 p. m.: Animal killed, one hour and twenty-four minutes after injection of trypan blue and air into left pleural cavity.

Forty-four minutes after injection of trypan blue and air into the left pleural cavity, blue appeared at the collecting cistern.

Total amount of lymph flow into cistern for thirty minutes before pneumothorax, 12.6 cc.

Total amount of lymph of flow into cistern for one hour, twenty-four minutes after pneumothorax, 17.2 cc.

Dog 6.—Jan. 4, 1928: 8:00 a. m.: Animal given regular morning meal.

10:59 a. m.: Flow of lymph from cannula in venous lymphatic cistern began; milky white.

11:27 a. m.: Total amount lymph collected, 41.1 cc.

11:29 a. m.: Two cubic centimeters of 1 per cent solution trypan blue and 250 cc. of air injected into the left pleural cavity. Breathing at once deepened and slowed. The flow of lymph was, as uniformly observed, noticeably less, and at

the same time what was extremely striking, the milkiness of the lymph at the cistern suddenly disappeared and the fluid became clear yellowish amber.

11:40 a. m.: Milky quality returning to lymph and flow, which had very greatly lessened immediately after the institution of the pneumothorax, was definitely increasing. The rate of flow was evidently less than before the pneumothorax.

12:24 p. m.: Animal killed, fifty-five minutes after the injection of trypan blue and air into the left pleural cavity. No blue in detectable quantities appeared in the collecting cistern during the entire experiment.

Total amount of lymph flow into cistern for twenty-eight minutes before pneumothorax, 41 cc.

Total amount of lymph flow into cistern fifty-seven minutes after pneumothorax, 38.9 cc.

PROTOCOLS

Autopsies were painstakingly performed in every case, and the observations have raised several interesting possibilities in regard to intrathoracic lymphatic drainage, but nothing germane to this particular problem was uncovered. We could see little difference in the actual amount of the residual trypan blue in the pleural cavities. The diffusion of the colloid material into the perilymphatic tissues seemed neither more nor less with or without pneumothorax. The rate of trypan blue absorption, even without artificial closed pneumothorax, was so gradual that it was impossible to detect any measurable difference in the amount of free trypan blue in the pleural cavities. We cannot see that a detailed description of the protocols could in any way aid in the interpretation of our actual experimental work, and we have, therefore, omitted them. It is well, however, to say that even in those animals in which no blue appeared at the collecting cistern during the life of the experiment, the same macroscopic involvement of the lymph node was apparent, i. e., the intertracheal-bronchial, and the left and right peritracheal bronchial groups. They were definitely enlarged, soft, edematous and blue.

SUMMARY OF EXPERIMENTS

A closed pneumothorax of 250 cc. produced in these animals two outstanding and arresting changes:

1. An average decrease of 46 per cent occurred in the rate of lymph flow at the venous lymphatic cistern.

2. Marked prolongation of the interval after the injection of trypan blue into the left pleural cavity was noted before any appreciable amount appeared in the collecting cistern.

INTERPRETATION OF RESULTS

In the lymph flow through the thoracic duct, the negative intrathoracic pressure incident on inspiration is of great importance, as has been already said, on the aspiration of lymph from the abdominal cavity into the intrathoracic portion of the duct. The increase of pressure

occurring with expiration is of just as great value in squeezing the lymph along toward and into the venous system at the angulus. The valves hold the gain made by inspiratory suction during expiratory pressures. Thus, under normal conditions, the efficient pulmonary pump delivers the lymph into the general circulation. Anything which limits the respiratory movements must necessarily influence, not only the lymph flow in the thoracic duct, but also to even greater extent, must affect the flow in those lymph vessels originating and having their entire course within the thorax itself. Man sleeps from five to nine hours daily; therefore during approximately two thirds of his life his body is in the vertical position, and in this posture the lymphatic collecting tubes must compete full against gravity. The pulmonary pump must of a necessity play an important rôle indeed during man's working hours in the delivery of lymph beyond the lymphatic venous valve.

When the respiratory pump is reduced in its efficiency, the chief remaining motive factor in lymph flow is the force built up from behind. But the force from behind must work now against the handicap of reduced pump performance, and the strain is referred back to the headwaters, that is, to the tissue spaces. Pressure in the tissue fluid must inevitably be raised. If this is slight, tissue interchange occurs, probably on a somewhat higher pressure level. Osmosis and filtration meet the emergency created by the inability of the lymph vessels to equilibrate local tension, and if tissue activity is sufficiently reduced no untoward results occur. But the maximum capacity of these emergency mechanisms may be overtaxed; the local balance is upset, lymph then accumulates, pressure in the lymph vessels increases since the chief factor in transporting it to the venous system is now the pressure from behind. Before the discharge of lymph at the angulus can be accomplished, local pressure in the tissue spaces rises to such a point that edema results. This edema, if of any extent, manifests itself in the pleural cavity as an effusion. This, we believe, is often the mechanism in the production of pleural effusion that occurs following a large artificial pneumothorax.

The production of a large pneumothorax experimentally was seen to result in a decrease of almost 50 per cent in lymph flow into the venous lymphatic cistern; it is probable that a considerable proportion of the lymph thereafter collected came from the abdominal cavity, as the following striking observation seems to verify.

In the single experiment in which the dog had inadvertently received his morning meal, the fat-laden, milky lymph from the digestive tract completely disappeared immediately on the production of the pneumothorax. The remaining lymph was clear amber and scanty. In ten minutes, the lacteal flow reappeared in the collecting cistern, and the rate of flow increased proportionately. It is evident then that momenta-

rily the abdominal lymph flow ceases altogether following a large artificial closed pneumothorax, but gradually there is a readjustment of pressure conditions in the tissues, and eventually, despite the changed respiratory movement, the equilibration of tissue pressure is accomplished and the threshhold actually requiring lymph flow in order to adjust tissue pressure is somewhat higher. Again it may be that the basal metabolism of the abdominal organs is less, thereby reducing absolutely the actual amount of molecular subdivision and of osmotic pull into the tissues. It is likely that both factors operate definitely to reduce the amount of lymph flowing from the abdominal cavity. So then, the decrease in the amount of lymph at the collecting cistern after the creation of an artificial pneumothorax represents probably a great reduction in the thoracic lymph flow, and some reduction at least in the abdominal lymph flow.

Dybkowsky,[2] Wadsworth,[3] Karsner and Swanbeck,[4] Costain,[5] Herring and MacNaughten,[6] Corper,[7] David [8] and many others [9] have shown by accurate and painstaking experimental work that various bacteria are removed under ordinary conditions almost exclusively by the lymph capillaries from the pleural and peritoneal cavities. MacCallum [10] conclusively demonstrated that phagocytes mustered out for the purpose, conveyed these bacteria into the lymph streams.

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2. Dybkowsky: Ueber Anspangung und Absonderung der Pleural-Wand, Arb. a. d. Phys. Anstalt, Leipsig **1**:40, 1866.

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10. MacCallum, W. G.: Mechanism Absorption of Granular Materials from Peritoneum, Bull. Johns Hopkins Hosp. **14**:146, 1903.

Grober [11] found that injection of bacteria alone into the pleural cavity was not attended by any untoward results. If, on the other hand, a chemical irritant was first injected between the pleural leaves, an empyema invariably resulted. He concludes that the integrity of the pleura in bacterial invasion depends on the power of the lymph vessels to remove bacteria to the main blood stream.

Noetzel [12] found that a twenty-four hour bacterial bouillon culture introduced into the pleural cavity without deleterious changes would cause empyema if an artificial pneumothorax was induced at the same time. The injected material not only remained but an exudation into the pleural cavity occurred and empyema resulted. He concluded that the pneumothorax, by reducing the respiratory activity, greatly interfered with the lymph and blood circulations. Just how he believed this mechanism operated, he did not state.

As bearing on this problem of the formation of an empyema, Allen [13] has recently shown that the presence of blood in the pleural cavity is an important factor in the production of empyema and that its speedy removal is the most efficient measure in avoiding this complication.

Tendeloo [14] believed that the lymph has a centroperipheral movement with inspiration and peripherocentral with expiration, and that the amount of thoracic lymph drainage is directly proportional to the amount of respiratory activity. He did not quote any experimental work, however, in defense of his conclusion on this point.

Graham,[15] in experimental work undertaken to study some of the factors in production of pleural exudates, found that the subpleural lymph vessels of the lung actually do become filled during inspiration, and that during expiration the fluid in these vessels is apparently squeezed out into the pleural cavity. This phenomenon can be best observed in an edematous lung. He concluded that this mechanism constitutes an important factor in the production of pleural exudates.

Nageli [16] stated that following thoracoplasty, patients uniformly and immediately felt much improved. He concluded that lymph stasis due

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11. Grober, H.: Die Resorptionskraft der Pleura, Habilitationsschrift, Jena, 1901.

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15. Graham, E. A.: Influence of Respiratory Movements on the Formation of Pleural Exudates, J. A. M. A. **76**:784 (March 19) 1921.

16. Nageli: The Changes of the Serological Reaction of the Blood after Extrapleural Thoracoplasty, Beitr. z. klin. Chir. **90**:351, 1914.

to the loss of the respiratory motive factor in lymph flow greatly reduces toxic absorption. He did not mention any corroborating experimental work.

Shingo,[17] working in Brauer's Clinic with rabbits and cats, found that after the inhalation of soot, the production of a unilateral pneumothorax greatly prolonged the time required for the elimination of the material on that side as compared with the unmolested thoracic cavity. He attributed this to the immobilizing effect of the pneumothorax on the respiratory movement, thus reducing lymph drainage.

And so these men and many since have concluded that thoracic lymph drainage is in a great measure dependent on the efficiency of the respiratory pump. As far as we can discover, however, the verification of the value of the respiratory movements as a motive force in lymph flow by actual measurement at the angulus venosus has never been done.

Bettman[18] found that with the simultaneous injection of india ink and air (from 20 to 50 cc.) into a pleural cavity, definite evidence of pleural reaction were obtained that were not present if india ink without artificial pneumothorax was employed.

Unquestionably, the physiologic circulatory balance in the pleural leaves constantly in apposition is a delicate one and an artificial pneumothorax of even 50 cc. must cause considerable lymphatic disturbance, although so little would scarcely be sufficient to affect materially the respiratory motive mechanism.

Lemon and Eloesser, in their discussion of Bettman's work, suggested the possibility that lymphatic blocking might occur following a closed artificial pneumothorax. Bettman concurred in this.

Marchand[19] stated many years ago that provided the lymph and blood streams are not interfered with, the injection of bacteria into the pleural space simply results in a temporary local reaction on the surface of the pleura, without actual injury to the pleural endothelium itself. If, however, there is a marked disturbance of lymph or blood circulation or if there is an initial injury to the pleural endothelium, empyema results.

Gardner,[20] in a very important study of the pathology of artificial pneumothorax, found that where the lymph trunks are the widest the inflammatory fibrosis is the greatest. He concluded that in continued

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17. Shingo: Beitrage zur Physiologie des kunstlichen Pneumothorax von seiner Wirkung auf die Lungentuberkulose, Beitr. f. klin. Tuberk. **11**:1, 1908.

18. Bettman, Ralph: Reaction Pleura to Particulate Matter: Especially in Presence of Pneumothorax, Arch. Surg. **10**:523 (Jan.) 1925.

19. Marchand: Zur Kenntnis der Fibrinosen Exsudation bei Entzundungen, Virchows Arch. f. path. Anat. **145**:279, 1896.

20. Gardner, Leroy V.: The Pathology of Artificial Pneumothorax in Pulmonary Tuberculosis, Am. Rev. Tuberc. **10**:501, 1924.

artificial pneumothorax, lymph stasis and retained metabolic products stimulate the formation of fibrous tissue and where the latter is most evident, in the peripheral portion of the lung, lymph stasis is also the greatest. Therefore, it would seem that the longer a pneumothorax continues, the more marked is the visceral pleural fibrosis and the higher the intrapulmonary pressure required to produce an intrapleural effusion.

CONCLUSIONS

Bilateral closed pneumothorax produced in dogs results in a great reduction of intrathoracic lymph flow. There seems little reason to doubt that this also occurs in the human being.

This may act: (*a*) beneficially in suppurative diseases of the lungs, by lessening toxic absorption that is breaking down or exhaustive resistance, (*b*) harmfully, by producing an effusion with its attendant danger of infection by pyogenic organisms.

The dramatic improvement felt by the patient after the institution of pneumothorax is due, we believe, to the reduction of lymph flow from the homolateral lung through the loss of its respiratory activity. For some time we have been accustomed to judge the efficiency of a closed pneumothorax in pulmonary tuberculosis by the temperature reaction. As long as the quantity of air in the pleural cavity is sufficient to prevent lymph flow from the diseased pulmonary tissue no toxemia occurs and there is therefore no elevation in temperature. When this transpires, however, more air is needed. The ultimate aim should be to constantly maintain the pressure just high enough to prevent the pulmonary lobes under treatment from participating in respiration.

Intelligent and careful pulmonary immobilizing procedures will be attended with the best results. Rest reduces toxemia by the "physiologic amputation" as John Alexander so tritely puts it, of the diseased lung. Gardner [20] has shown that lymph stasis results, thereby stimulating fibrosis, the nemesis of the tubercle bacilli. Rest is the best treatment for tuberculosis, but is it not the lymph stasis, the result of rest, causing fibroplastic proliferation that actually defeats the tubercle bacilli?

To create a closed pneumothorax that just rests the involved pulmonary tissue and to maintain this comparative immobilization should be the goal sought where indication for pneumothorax is clearly cut. The less lung sacrificed by the institution of a closed pneumothorax, the less compensatory activity of the remaining functioning tissue. The collapse of an entire lung frequently breaks down the barriers that have been erected against tuberculous foci harbored in the contralateral lung.

The same principle applies to thoracoplasty when the indication for marked collapse does not require extensive resections of the ribs.

The operation of choice, it seems to us, is the one that gives the maximum amount of rest and lymph stasis, and therefore fibrosis with the minimum amount of pulmonary collapse and circulatory disturbance.

ABSTRACT OF DISCUSSION

DR. RALPH B. BETTMAN: Anybody who has ever attempted the collection of lymph from the thoracic duct will appreciate what a difficult task Dr. Dolley has performed. In my work, which Dr. Dolley quoted, I attempted this collection of lymph to study the time of appearance of the india ink which had been injected into the pleura. I met with so many technical difficulties, and my results seemed so uncertain, that in the paper I made no reference to that part of the experiment. Many factors are involved in the absorption of material from the pleural space in the presence of a pneumothorax. Of course, the fact that the lung has been collapsed and therefore the pleural surface area available has been diminished, accounts to a certain extent for the diminution which I found in the absorption from particles of india ink. There are other factors involved, however, as I attempted to show at that time. Many of my specimens showed that large quantities of india ink were apparently blocked in the pleura and the immediately adjacent lung tissue.

Dr. Dolley's results are extremely interesting, and throw a new light on this complicated problem.

DR. JOHN ALEXANDER: There are two clinical observations that tend to confirm Dr. Dolley's experimental conclusions. The first is that sometimes in pulmonary tuberculosis great improvement follows phrenicectomy that, because of pleural adhesions or an unusually firm lung, fails to cause a satisfactory rise of the diaphragm. The favorable effect here is, I believe, due to the pulmonary rest that follows diaphragmatic paralysis. The second is the remarkable improvement that occurred in two tuberculous patients following phrenicectomy and later resection of posterior portions of ten intercostal nerves. In these patients not only the diaphragm but the wall of the chest, and in turn the lung, became virtually immobile.

I mention these cases in connection with Dr. Dolley's paper, because the improvement seems to have been the result of thoracic and pulmonary rest alone rather than the rest and relaxation or compression that follows a thoracoplasty or a phrenicectomy with marked rise of the diaphragm.

DR. DOLLEY: I agree with Dr. Bettman with regard to the interpretation of his results. It is rather uncertain just how much air was present in the pleural space with the manipulation going on at that time, though probably it was a respiratory motive mechanism that was interfered with.

Dr. Alexander's point seems good, that further reduction in lung activity on the side under experimentation might still more retard lymph flow. We contemplate going on with that work and, after a phrenicectomy, finding out what results may occur in regard to flow at the lymphatic venous angle.

THE "STRAIN" ON THE COLLATERAL LUNG IN COLLAPSE THERAPY *

EDWARD D. CHURCHILL, M.D.
BOSTON

In the lung, as in nearly every organ of the body, there exists a large margin of functional reserve which may be called into play when conditions demand. It is this reserve that is utilized when one lung is required to take over the work of both, either because of an extensive pathologic process or as a result of the collapse of one lung induced as a therapeutic measure to afford functional rest. It is commonly stated that the production of even a partial collapse of a diseased lung throws a "strain" on the collateral side, and it is almost as universally accepted and feared that this added strain may in some way favor the spread of the disease to the sound side, or at least may tend to fan into activity a smoldering or quiescent focus of disease.

It is on the basis of such reasoning that the condition of the collateral lung assumes such importance in the selection of cases of tuberculosis for thoracoplasty; and it is this conception which is held in mind when a preliminary section of the phrenic nerve is advised as a minimum procedure, designed to test the ability of the opposite lung to withstand the "strain" which is to be put on it by further and more radical measures.

An attempt is being made, therefore, to analyze the mechanisms by which a lung responds to a demand for increased function. When these are understood, the way may be opened for a consideration of whether compensatory activity actually does make the lung a more favorable soil for the implantation of a new, or the reactivation of an old tuberculosis.

The compensatory adjustments of the organism as a whole to a reduction of lung volume have been determined by Heuer and Andrus [1] in studies concerning the effects of pneumectomy. Briefly, they found that "for about thirty days after operation there is a temporary increase in the alveolar CO_2, a fall in the alveolar oxygen, a slight increase in the CO_2 of the blood, and a marked decrease in the blood oxygen. By way of compensating for these changes until the enlargement of the remaining lung shall have taken place, the number of red blood cells, the hemoglobin and, consequently, the oxygen-carrying capacity of the blood are increased." In addition, Andrus [2] found "a transient increase in the pulse rate and the blood flow through the remaining lung persist-

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* From the West Surgical Service of the Massachusetts General Hospital.

1. Heuer, G. J., and Andrus, W. D. W.: Bull. Johns Hopkins Hosp. **33**:130 (April) 1922.

2. Andrus, W. D. W.: Bull. Johns Hopkins Hosp. **34**:119 (April) 1923.

ing for about ten days, followed in both by a fall slightly below the preoperative value."

With reference to adjustments taking place within the lung itself, studies have centered about the dynamics of the pulmonary circulation. A number of experimenters (Wiggers,[3] Underhill,[4] Haggart and Walker[5]) have measured the increase in pressure in the pulmonary artery which is produced by the occlusion of one of its main branches. Recently, Scarff[6] has measured the pressure in the pulmonary artery of dogs during an interval of fourteen days subsequent to the operation of pneumectomy. It was found that there was an immediate increase of from 30 to 50 per cent in the systolic pressure of the pulmonary artery —figures which agree with the usual observations—and an increase of from 35 to 100 per cent in the pulse pressure. This increase persists for a number of days but eventually returns to normal, the time required being between seven and twenty-one days.

A similar increase in the pressure of the pulmonary artery may be observed when the return flow of blood to the right side of the heart is increased. If in a heart-lung preparation the volume of blood poured into the right side of the heart is increased or decreased, the pressure in the pulmonary artery rises or falls in direct proportion. It is highly probable, therefore, that in exercise, in which there is at times a fourfold increase in the volume output of the heart (Bock and co-workers[7]), there is a notable increase in the pressure in the pulmonary artery. The increased consumption of oxygen attending exercise also calls for an increase in the functional activity of the lungs, and with exercise—as well as under the conditions produced by occluding a portion of the stream-bed—one looks for a compensatory increase in the diffusing surface of the lung.

The obvious and immediate response of the respiratory system is an increase in ventilation by a modification of the rate and depth of breathing. This increase in ventilation, however, is not in itself sufficient to meet the demands of severe exercise, and the question is asked whether the altered dynamics of the pulmonary circulation, as evidenced by the rise in pressure in the pulmonary artery may not act directly to increase the functional diffusing surface. The following experiments appear to answer this question in the affirmative.

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3. Wiggers, C. J.: Physiol. Rev. **1**:239 (April) 1921.

4. Underhill, S. W. F.: Brit. M. J. **2**:779, 1921.

5. Haggart, G. E., and Walker, A. M.: Physiology of Pulmonary Embolism as Disclosed by Quantitative Occlusion of Pulmonary Artery, Arch. Surg. **6**:764 (May) 1923.

6. Scarff, John E.: Pulmonary Blood Pressure, Arch. Surg. **12**:591 (Feb.) 1926.

7. Bock, A. V., and others: Personal communications.

METHODS AND EXPERIMENTS

A cat is completely anesthetized by an intravenous injection of sodium barbital (5 per cent solution) and prepared in the manner described by Churchill and Agassiz,[8] which permits the separation of the air breathed by the two lungs. In brief, this is accomplished by tying the end of a long cannula in the left primary bronchus and then making an air-tight closure of the chest by suturing the pericardium to the edges of a window made in the anterior wall of the chest. In order to secure a continuous record of the oxygen consumption and the respiratory minute-volume, the airway leading from each lung is connected with a closed spirometer system consisting of a tube of soda-lime and a Brodie bellows—the latter writing directly on the kymograph. The animal breathes an atmosphere of pure oxygen as in the familiar graphic metabolism apparatus.

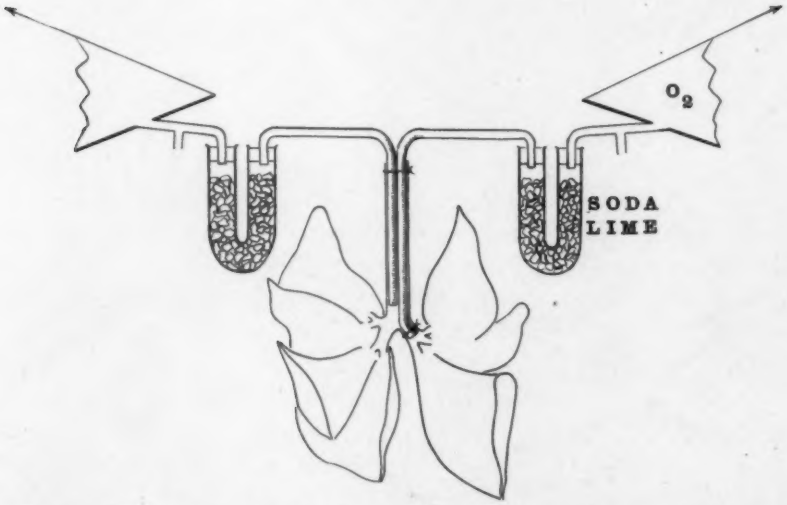

Fig. 1.—The position of the cannula in the left lung is secured by a ligature around the left primary bronchus. The spirometer system is based on the principle of a graphic metabolism apparatus.

In order to increase the functional demand on one lung, the branch of the pulmonary artery to the opposite side is suddenly occluded by compression with a ligature previously placed about the vessel. This shunts the entire output of the right ventricle into one lung, thereby more than doubling the volume of flow in the left lung when the right pulmonary artery is shut off. A similar diversion of the volume of flow may also be produced by occluding either set of pulmonary veins. The placement of the ligatures about these vessels within the pericardium is made relatively simple by the exposure of the heart which this preparation affords.

EXPERIMENT 1.—A cat weighing 3 Kg. was prepared in the foregoing manner. Figure 2 represents a portion of the kymograph record obtained from this animal. The upper tracing is that of the systemic blood pressure measured in the carotid artery in millimeters of mercury. Below the base line of the blood pressure

8. Churchill, E. D., and Agassiz, Anna: Am. J. Physiol. **76**:6 (March) 1926.

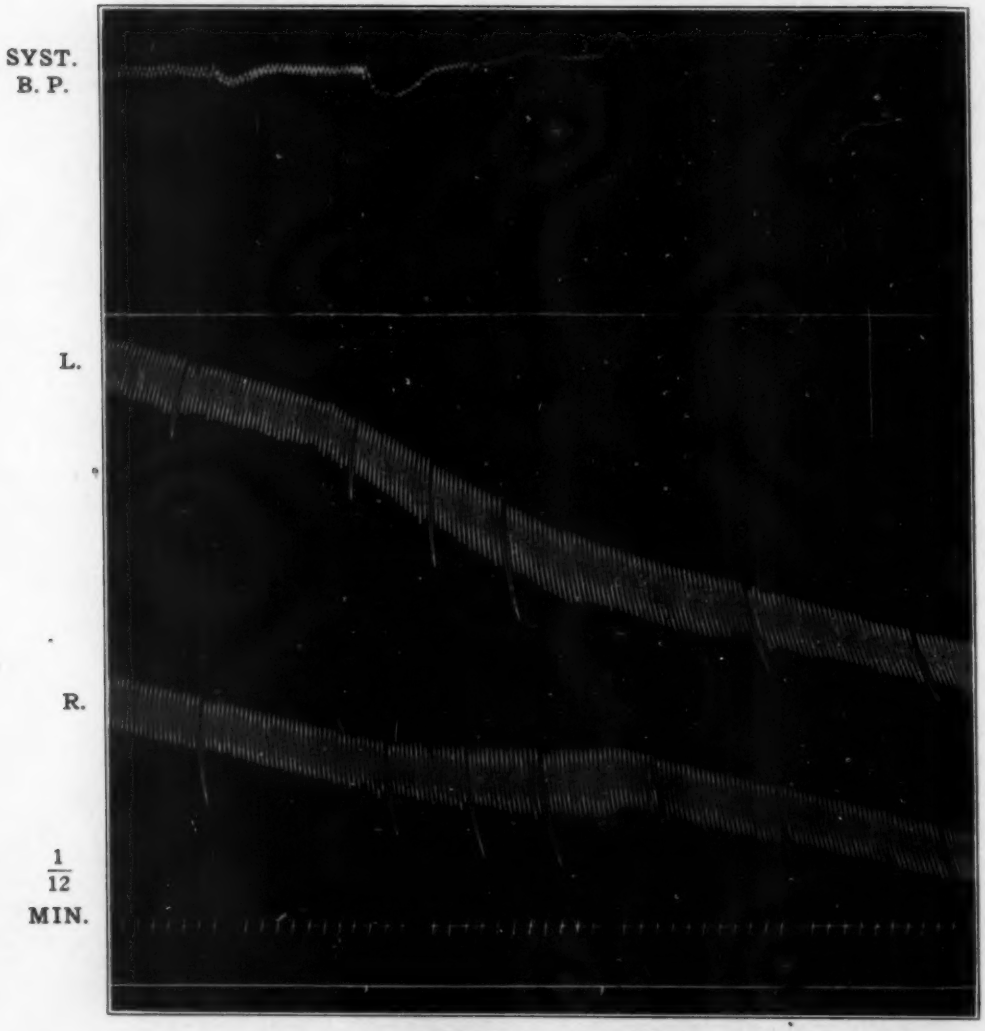

Fig. 2 (experiment 1).—The systemic blood pressure is measured in millimeters of mercury from the base line. The respiratory tracings of the two lungs are recorded by the bellows, the tracing of the left lung being uppermost. Oxygen consumption is determined by the pitch of the tracing as measured from the lower base line. The signal marker on the base line has recorded the points at which the right pulmonary artery was occluded and subsequently released.

appear the tracings of the two Brodie spirometers, that connected with the left lung being uppermost. These are so calibrated that the volume of each breath can be measured in cubic centimeters and the total minute-volume of ventilation computed. The slow fall in the level of the writing point, as the tracing is read from left to right, is an index of the oxygen consumption of the animal, and its value can be translated to cubic centimeters per minute by measuring from the base line. The time marker records one-twelfth minute intervals.

The point at which the right pulmonary artery was occluded is marked by a flattening of the curve traced by the right lung, indicating the abrupt cessation of oxygen absorption by that lung. The curve traced by the left lung, however, assumes a steeper pitch as this side takes over the function of the right. The numerical values computed from this experiment are shown in table 1, and from a study of this table one is able to determine the manner in which the left lung accomplishes this sudden increase in function.

With the first occlusion, it is to be noted that although the total ventilation is increased by 40 cc. (2.4 per cent), the minute-volume of the left lung remains

TABLE 1.—*Data on Experiment 1*

Period	Respiration Rate per Minute	Volume per Breath		Minute-Volume		Total Minute-Volume	Oxygen Absorption		Total Oxygen Absorption	O_2 Absorption Min. Volume	
		L	R	L	R		L	R		L	R
Control.....	45	17.5	18.5	788	835	1,623	15.0	21.0	36.0	0.018	0.024
Right pulmonary artery occluded	44	17.9	19.9	788	875	1,663	28.8	0.0	28.8	0.036	0.0
Control.....	45	18.9	19.1	853	859	1,712	16.8	18.6	35.4	0.019	0.021
Control.....	41	13.6	18.6	558	766	1,324	13.0	16.0	29.0	0.023	0.029
Right pulmonary artery occluded	40	18.9	21.7	756	869	1,625	28.0	0.0	28.0	0.037	0.0
Control.....	38	17.9	18.7	683	711	1,394	14.0	15.0	29.0	0.020	0.021

unchanged. Nevertheless, the oxygen absorbed by this lung during this period increases from 15 to 28.8 cc.—an increase of nearly 50 per cent. It is unusual in the course of these experiments to find this absolute lack of an increase in ventilation, but this one incident shows that under the proper conditions it may occur.

The usual response, as just stated, is for the animal to increase definitely the minute-volume ventilation, the change being shared equally by the two lungs. To show that the compensatory absorption of oxygen is not merely a result of this increased ventilation, the final two columns of the table have been computed to show the ratio between the oxygen absorption and the minute-volume. This decimal fraction in a manner expresses the effectiveness of the diffusing surface; that is, the rate of oxygen absorption as compared with the ventilation under the altered conditions of blood flow. The figures consistently show an increase in the effectiveness of the diffusing surface when the circulation through the lung is increased.

EXPERIMENT 2.—The results of a similar experiment are shown in table 2. In addition, the pressure in the main stem of the pulmonary artery was measured. This was recorded by the technic used by Haggart and Walker,[6] with the addition of a water manometer on the far side of the membrane tambour. At the point of occlusion of the right branch, the pressure in the pulmonary artery rose abruptly from 18 to 23 cm. of water, or an increase of 28 per cent.

The procedure described in these two experiments has been repeatedly demonstrated in some fifteen different animals with consistent results. The same effect is observed when stasis of the circulation in one lung is brought about by occlusion of its venous return.

COMMENT

These experiments have demonstrated that the burden thrown on a lung by a sudden increase in volume of flow is not altogether borne by increasing the ventilation, but is in part compensated for by an increase in the area of the functional diffusing surface. One of the ways in which this may be brought about is by the opening of reserve capillary pathways; in respect to this mechanism, the observations of Wearn [9] are of notable interest.

This author and his co-workers reported, on the basis of direct microscopic evidence, that the capillaries around a single alveolus may appear and disappear from time to time. Such an opening of new

TABLE 2.—*Data on Experiment 2*

Period	Respiration Rate per Minute	Volume per Breath		Minute-Volume		Total Minute-Volume	Oxygen Absorption		Total Oxygen Absorption	O_2 Absorption Min. Volume	
		L	R	L	R		L	R		L	R
Control.....	18	16	13	288	237	525	12.0	11.0	23.0	0.041	0.046
Right pulmonary artery occluded	21	17	14	356	302	658	22.0	0.0	22.0	0.061	0.0
Control.....	22	17	13	304	286	650	12.5	11.5	24.0	0.034	0.040
Right pulmonary artery occluded	22	18	15	386	325	711	22.5	0.0	22.5	0.058	0.0
Control.....	21	17	14	351	298	649	11.0	12.5	23.5	0.031	0.041

vascular pathways, in response to the increased head of pressure in the pulmonary artery, is consistent with the observation that the blood content of the lungs increases when the rate of flow is increased (Drinker, Churchill and Ferry [10]).

In view of this evidence, which demonstrates one more way in which the organism is able to respond to a demand for increased respiratory function, one is again led seriously to question what is meant when reference is made to the "strain" on the collateral lung. Under conditions in which the aeration of a portion of the lung is reduced, but the flow of blood through it maintained, the return of the fraction of unarterialized blood to the left side of the heart and general circulation may lead to a marked increase in the rate and depth of breathing. Hofbauer [11] felt that such exertion may cause a liberation of tuberculin and

9. Wearn, J. T.: Barr, J. S., and German, W. T.: Proc. Soc. Exper. Biol. & Med. **24**:114 (Nov.) 1926.

10. Drinker, C. K.; Churchill, E. D., and Ferry, R. M.: Am. J. Physiol. **77**:590 (Aug.) 1926.

11. Hofbauer, Ludwig: Handbuch der Normalen und pathologischen Physiologie, Berlin, Julius Springer, vol. 2, 1925, p. 408.

produce a reaction by "auto-tuberculinization." This author also expressed the belief that a relative high negative pressure on the side of the sound lung may actually suck infectious lymph into that side. Whether either of these suggestions can be proved or not, it seems probable that if a harmful strain is ever thrown on the collateral lung it is most likely to be through this increase in the rate or depth of breathing. This, as has been stated, is more apt to result from the return of unarterialized blood to the left side of the heart than as a compensatory measure on the part of the sound lung to carry on the functions of respiration when a larger flow of blood is diverted to it.

If this conception of the pathologic physiology is kept in mind when clinical cases are studied with view to collapse therapy, it becomes apparent that many times one may greatly discount the item of a possible "strain" on the collateral lung arising from an increased functional burden. In other words, it is possible that a strain may arise from the compensatory breathing when portions of lung are collapsed but continue, at least for some time, to be circulated actively. The blood from such an area is returned to the general circulation low in oxygen and with a high content of carbon dioxide, and acts as a respiratory stimulant. In contrast, however, the strain arising from the simple diversion of blood to the opposite lung is presumably minimal. Procedures involving the collapse of the functioning lung therefore are to be avoided, and when necessary should be carried out in several stages.

This consideration has been confined to the question of "strain" in relation to compensatory pulmonary function. It is obvious that in clinical surgery one can never discount the general effects of a surgical operation, with its attendant shock and loss of blood, in a debilitated patient.

ABSTRACT OF DISCUSSION

Dr. Edward N. Packard: I should like to ask Dr. Churchill whether in artificial collapse of the lung there is any occlusion of the arteries or veins. A difference of opinion exists as to whether the blood flow through the lung is increased or decreased after the lung is collapsed. A variation in the flow of blood through the collapsed lung may or may not have any effect on the state of the tuberculosis in the opposite lung, but clinically in the majority of cases there is a clearing up of the disease in the so-called good lung after a satisfactory collapse of the bad lung.

Dr. W. D. Andrus: I want to congratulate Dr. Churchill on this investigation. I was much interested in his method of obtaining the separate respiration of the two lungs, and I think he has there indirect evidence of the actual volume of the lung. No doubt he has the idea in mind of actually determining the volume of the lung, and I would like, if he has not already done so, to suggest that he add a certain percentage of nitrogen to the bellows, and then by subsequent analysis of the oxygen content of the bellows, he will be able to estimate the actual volume of those two lungs.

With regard to the matter of circulation of the other lung, I should like to hark back to something we reported—a method. We simply performed a ligation of the bronchus, placing about the pulmonary artery a clamp to make subsequent compressions of the artery, and by that means we found through estimating blood volume from oxygen determinations and the oxygen absorption, that something like 30 per cent of the total blood flow, at least 30 per cent, still went through the collapsed lung. We used bronchial ligations as the nearest possible means of simulating conditions of pneumothorax in a dog.

DR. EDWARD CHURCHILL: I think that Dr. Andrus has answered Dr. Packard's question much more ably than I could because I should have cited Dr. Andrus' work on that subject.

I thank Dr. Andrus for his suggestion. We all know it is difficult to obtain a vital capacity reading in animals, but I hope to continue with the study of this problem.

PHRENICOTOMY IN THE TREATMENT OF DIAPHRAGMATIC HERNIA AND OF TUMORS OF THE WALL OF THE CHEST

STUART W. HARRINGTON, M.D.
From the Division of Surgery, Mayo Clinic
ROCHESTER, MINN.

Induced paralysis of the diaphragm by cervical phrenicotomy as a method of treatment of certain severe chronic pulmonary lesions was first suggested by Stuertz [1] in 1911. The proposal of Stuertz followed experimental work of Senac, Arnemann, Purkinje, Budge and Gerhardt,[2] and was made on the basis that the diseased lung would be helped in the sense of collapse therapy by immobilization and compression of the diseased tissue. In 1912, Hellin [3] differed from this view and maintained that the diaphragm never becomes actually immobilized. He stated that each half of the diaphragm is innervated not only by the corresponding phrenic nerve, but by bundles arising from the intercostal nerves, and that the phrenic nerves anastomose with each other. After experimental work on guinea-pigs, rabbits and dogs, he stated that section of the phrenic nerve does not always cause paralysis of the diaphragm. Therapeutic paralysis of the diaphragm was first effected in man at the suggestion of Stuertz by Bardenheuer,[4] in 1912, in a case of bronchiectasis of the lower lobe of the left lung. In 1913, Sauerbruch [5] published reports of five cases. Friedrich [6] reported eight cases in 1914, and Walther,[7] in the same year, reported twenty-six from Sauerbruch's clinic.

The procedure has been used mainly in the treatment of pulmonary tuberculosis and to some extent in nontuberculous pulmonary infections.

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1. Stuertz: Künstliche Zwerchfellähmung bei schweren chronischen einseitigen Lungenerkrankungen, Deutsche med. Wchnschr. **2**:2224, 1911.

2. Senac; Arnemann; Purkinje; Budge; Gerhardt, quoted by Lange: Deutsche Ztschr. f. Chir. **169**:199, 1922.

3. Hellin, Dionys: Ueber Zwerchfellähmung nachinseitiger Phrenicusdurchschneidung, Deutsche med. Wchnschr. **2**:1460, 1912; Zur Geschichte der Phrenikotomie, München. med. Wchnschr. **2**:1888, 1925.

4. Bardenheuer, quoted by Lange.

5. Sauerbruch, Ferdinand: Die Beeinflussung von Lungenerkrankungen durch künstliche Lähmmung des Zwerchfells (Phrenikotomie), München. med. Wchnschr. **1**:625, 1913.

6. Friedrich, P. L.: Die operative Methodik bei der chirurgischen Behandlung der Lugentuberkulose durch Rippenabtragung, operative Phrenicus und Intercostalnerven-Lähmung, Verhandl. d. deutsch. Gessellsch. f. Chir. **43**:254, 1914.

7. Walther, H. E.: Röntgenologische Untersuchungen über die Wirkung der Phrenikotomie, Beitr. z. klin. Chir. **90**:358, 1914.

In such cases it is rarely a curative procedure in itself, but usually auxiliary to other forms of treatment and often serves as a test of the patient's ability to withstand more radical surgical procedures. In many cases of early disease of the lower lobe the symptoms are entirely relieved. The diseases for which this procedure has been used have gradually increased to include not only pulmonary lesions but lesions of the pleura and diaphragm. It is also used in the treatment of symptoms in which the motion of the diaphragm plays an important rôle.

In cases of chronic empyema in which narrowing of the chest cavity is desired, the relaxed diaphragm assists in collapsing the cavity and lessens the magnitude of subsequent operations to produce complete obliteration. In 1920, Sauerbruch [8] recommended its use in combination with extensive plastic operations on the wall of the chest. In 1922, Lange [9] reported a case of empyema in which there was a good result. I have secured favorable results in cases of tuberculous and nontuberculous empyema in which it was necessary to collapse extensive cavities by open operation. I have also found it of great aid in obliterating extensive empyema cavities associated with extrapleural thoracoplasty, and a subsequent open operation was not necessary to complete the obliteration of the cavity.

Davies [10] has stated that cardiac embarrassment resulting from extreme fibrosis of the lung or from pleural pericardial thickening and adhesions to the diaphragm may be improved by paralyzing the diaphragm, which will allow the heart to return to its normal position. Sauerbruch has stated that it is useful in pericarditis and adhesions of the heart, lung and diaphragm and that it has been used in cardiolysis.

Sauerbruch succeeded in narrowing the residual cavity one-third in a case of mediastinal cyst by phrenicotomy after removal of the cyst.

Diaphragmatic pleurisy with intolerable pain in the shoulder has been relieved by phrenicotomy, as reported by Lange who also quoted Oehlecker.[11]

In the syndrome of tonic contraction of the diaphragm, described by Duchenne,[12] it is suggested by Lange that relief will be obtained by bilateral phrenicotomy.

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8. Sauerbruch, Ferdinand: Die Chirurgie der Brustorgane, Berlin, J. Springer, 1920, vol. 1, p. 931; Die Chirurgie der Brustorgane, Berlin, J. Springer, 1925, vol. 2, p. 1075.

9. Lange, Kurt: Ueber pathologische und therapeutische Zwerchfellähmung, Deutsche Ztschr. f. Chir. **169**:199, 1922.

10. Davies, H. M.: Surgery in the Treatment of Pulmonary Tuberculosis, Brit. M. J. **2**:1145, 1924; Phrenic Evulsion as an aid in the Treatment of Pulmonary Tuberculosis and Bronchiectasis, ibid. **1**:315, 1926.

11. Oehlecker, F.: Zur Klinik und Chirurgie des Nervus phrenicus, Zentralbl. f. Chir. **1**:825, 1913.

12. Duchenne, C. B., quoted by Lange.

In 1914, Sauerbruch performed bilateral phrenicotomy in a case of tetanus with a favorable result.

Ghose [13] reported a case of hiccup relieved by temporary paralysis produced by injection of both phrenic nerves with procaine hydrochloride. Lilienthal [14] reported a case of obstinate hiccup which was completely relieved by injection of the phrenic nerve with alcohol. Dowman [15] reported a case of diaphragmatic tic following encephalitis which was completely relieved by section of both phrenic nerves.

Sauerbruch stated that coughing is made easier and expectoration freer by phrenicotomy. This view is also held by Davies, who stated that when the lung is adherent to the diaphragm an intolerant irritating cough is set up and the pumping action of the diaphragm during coughing may produce vomiting. He has used phrenicotomy in these cases since 1914 with good results and believes it should receive more consideration.

Warner [16] reported a case of injury to the chest with severe hemorrhage from the lung into the pleural cavity which stopped after the lung had been put at rest by phrenicotomy.

Sauerbruch has employed the procedure before resecting carcinoma of the cardia, and in experimental work on animals, in 1904, he developed technical problems associated with resection of the esophagus. He stated that in resection of the esophagus a flap can be made from the central tendon to reinforce the suture line of the esophagus. Lilienthal suggested thoracic phrenicotomy as a possible prophylaxis against hiccup, vomiting and coughing after mediastinal and transpleural esophagogastrostomy for stricture or carcinoma of the esophagus.

Lange stated that phrenicotomy is useful in the treatment of wounds of the diaphragm as they arrest the movement and diminish the tonus of the diaphragm, making suturing easy. Sauerbruch also advocated its use in these cases.

According to Lange, the paralyzed diaphragm may be used effectively under certain circumstances to close large defects of the wall of the chest. Sauerbruch has used it in a case of sarcoma "the size of a man's fist" originating in the sixth rib. He resected 10 cm. of the fifth, sixth and seventh ribs, and performed intrathoracic phrenicotomy.

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13. Ghose, D. M.: A Case of Persistent Hiccough Treated Successfully by Injections of Novocaine into the Phrenic Nerve, Indian Med. Gaz. **61**:124, 1926.

14. Lilienthal, Howard: Thoracic Surgery, Philadelphia, W. B. Saunders Company, 1925, vol. 2, p. 511-512.

15. Dowman, C. E.: Relief of Diaphragmatic Tic, Following Encephalitis, by Section of Phrenic Nerve: Report of a Case, J. A. M. A. **88**:95 (Jan. 8) 1927.

16. Warner, W. P.: Phrenicotomy in Traumatic Injury to Chest, Canad. M. A. J. **16**: 427, 1926.

The paralyzed diaphragm was then sutured to the upper edge of the cavity after the lung had been fixed to the wall of the chest. In this paper I am reporting two similar cases in which satisfactory results were obtained, and in one case a portion of the diaphragm was removed with the involved portion of the wall of the chest.

Last year I reported [17] a case of diaphragmatic hernia in which relaxation of the diaphragm was necessary in order to close the large traumatic hernial opening. Since reporting this case, I have found that Goetze,[18] in 1925, in reporting an article on the technic of radical phrenicotomy, mentioned two cases of traumatic hernia in which, in 1920, he had performed cervical unilateral phrenic emphraxis by freezing the phrenic nerve. In one of these cases he did not obtain complete paralysis of the diaphragm and this stimulated his interest in the anatomy and physiology of the phrenic nerve.

Sauerbruch suggested intrathoracic phrenicotomy, just above the diaphragm at the same time that the obstructing muscle ring is severed, in cases of strangulated diaphragmatic hernia. He stated that large hernial openings can be closed after phrenicotomy by suture, but if the muscle is hard and inelastic because of chronic fibrous inflammation with adhesions to the adjacent organs, phrenicotomy is useless. In these cases plastic methods are used in order to close the opening in the diaphragm. He stated that omentum is insufficient and that tissue of the liver, spleen, stomach and lung may be considered, but that fascia is most often used. He strongly recommended the transpleural operation in the repair of diaphragmatic hernia, and stated that of four cases of diaphragmatic hernia in which operation was performed in his clinic, the transpleural approach was used in three. In one case the operation was started by the abdominal approach, but was completed by thoracotomy because of the difficulty encountered in reducing the hernia. The patient died two days later from peritonitis. The other three patients recovered. He stated that these results are more satisfactory than statistics reported by Schumacher,[19] who reviewed fifty-six cases. Thoracotomy was performed in eleven cases and was successful in eight. Forty-five operations were performed by the abdominal approach with thirty-six deaths and nine recoveries. In only five of these nine were radical operations performed with closure of the hernial opening. This incidence of failure to complete the closure of the opening in the diaphragm and the mortality following abdominal opera-

17. Harrington, S. W.: Diaphragmatic Hernia, Arch. Surg. **16**:386 (Jan.) 1928.

18. Goetze, Otto: Die effektive Blockade des Nervus phrenicus (radikale Phrenicotomie), Arch. f. klin. Chir. **134**:595, 1925.

19. Schumacher, Oskar: Zur Entstehung der traumatischen Zwerchfellshernie, Arch. f. klin. Chir. **129**:782, 1924.

tion for diaphragmatic hernia is much higher than has been my experience. In the last three and a half years I have operated in fourteen cases of diaphragmatic hernia by the abdominal approach with closure of the opening in every case. Two deaths resulted. One patient died on the twelfth day from cardiorenal disease and cardiac failure. The other died of bronchopneumonia on the fourth day (case 5 reported in this paper).

Much of the recent literature deals with the anatomic structures of the phrenic nerve, the technic of methods of operation and the physiologic effect of paralysis of the diaphragm. It is not my object to discuss here the relative merits of the different operative procedures that have been advised and the differences of opinion as to the physiologic effects in the various conditions. From a technical operative standpoint it is important to recognize the variations in the anatomic structure and relations of the nerve. In cases in which complete and permanent paralysis of the diaphragm was desired, I performed phrenic exercise as described by Felix,[20] with satisfactory results. In cases in which temporary paralysis was desired I obtained good results by injecting or crushing the nerve. In all cases the function of the diaphragm is determined immediately by the use of the fluoroscope. In utilizing these procedures on the phrenic nerve in the treatment of diaphragmatic hernia it is important to know that there is no serious physiologic effect on respiration, or on the structures contained within the thorax or abdomen, and according to investigations the effect of permanent paralysis of the diaphragm on the function of important thoracic and abdominal organs is harmless. The physiologic effect on the healing of the diaphragm has not been definitely determined. I am now conducting experimental work on animals to determine this effect. Clinical observation of the patients on whom I have performed this operation indicates satisfactory healing, in that the hernia has not recurred two and a half years after operation.

ANATOMY

The phrenic is a mixed nerve, arising from the third, fourth and fifth (mainly fourth) cervical nerve roots. It passes downward and mesially across the anterior surface of the anterior scalene muscle. It is lateral to the thyrocervical trunk, inferior thyroid and ascending cervical arteries and vagus nerve and posterior to the transverse cervical and transverse scapular arteries. Before entering the thorax it passes between the subclavian vein and artery and behind the subclavius muscle at about the sternoclavicular articulation and crosses the internal mammary artery close to its origin. It passes through the middle medi-

20. Felix, Willy: Die Phrenicus-Ausschaltung bei Lungenerkrankungen, Ergebn. d. Chir. u. Orthop. **18**:690, 1925.

astinum beneath the mediastinal pleura accompanied by the pericardiacophrenic branch of the internal mammary artery and in front of the root of the lung to the pericardium and beneath the pericardial pleura to the diaphragm. The relations of the two pleural nerves differ in the thoracic course. The course of the right nerve is more vertical and lies lateral to the innominate vein and superior vena cava. It is shorter than the left nerve, more deeply situated, and enters the diaphragm in front of and lateral to the foramen venae cavae. The left phrenic nerve is crossed by the thoracic duct in the root of the neck. In the superior mediastinum it lies between the left common carotid and left subclavian arteries and crosses superficially to the vagus nerve on the left side of the arch of the aorta. It passes more ventrally to the root of the lung than the right nerve and enters the diaphragm more laterally and anteriorly.

The left phrenic nerve communicates with the ganglion of the inferior cervical sympathetics and often receives fibers from the nerve to the subclavian muscle and occasionally fibers from the ansa hypoglossi. Each nerve supplies filaments to the pericardium and pleura. Bands have been described passing to the peritoneum. The terminal branches pierce the muscle to which it is the chief if not the only motor supply, and to the greater portion of the abdominal and thoracic pleura of which it is the sensory supply.

The right nerve joins, by small filaments, in a small phrenic ganglion with phrenic branches of the celiac plexus; branches from the ganglion are distributed to the falciform and coronary ligaments of the liver, suprarenal gland, inferior vena cava and right atrium. From the left, nerve filaments pass directly to the celiac plexus without a ganglion and give filaments to the left suprarenal gland.

The sensory distribution to the diaphragm has not been definitely established. In 1853, Luschka [21] believed that the phrenic nerve gave off sensory fibers to the pleural surface of the diaphragm as well as motor fibers to the muscle. Poirier and Charpy,[22] and others, confirmed this opinion. Cavalié [23] has demonstrated that the last six intercostal nerves penetrate from 1 to 2 cm. into the margin of the diaphragm, supply the muscle with motor, and probably also contribute sensory, fibers to the pleural covering. Eisler [24] expressed the belief that all of the intercostal fibers to the diaphragm are sensory. Von Gössnitz [25] claimed that he could trace bundles of intercostal sensory nerves in the diaphragmatic pleura but could find only short motor branches. Ran-

21. Luschka, quoted by Capps, J. A.: An Experimental Study of the Pain Sense in the Pleural Membranes, Arch. Int. Med. **8**:717 (Dec.) 1911.
22. Poirier and Charpy (footnote 21).
23. Cavalié (footnote 21).
24. Eisler (footnote 21).
25. Von Gössnitz (footnote 21).

ström [26] concluded that the phrenic nerve furnishes sensory branches to the entire pleural covering of the diaphragm except at the rim where both pleura and peritoneum receive sensory fibers from the last six intercostal nerves.

Ferguson's [27] experimental work on cats, in 1891, proved the presence of sensory fibers in the phrenic nerve. He called attention to sensory disturbances in inflammatory conditions involving the diaphragm and reported a case of abscess of the liver with referred pain to the back of the neck and to the shoulder.

In 1911, Capps [28] observed clinically 240 cases of pleural pneumonia and pleurisy. He performed thoracentesis in thirty-five cases, exploring the diaphragmatic pleura with a wire through a cannula, and concluded that the diaphragmatic pleura derives its sensory supply from two sources, the phrenic nerve and the last six intercostal nerves. The central portion of the diaphragmatic pleura is innervated by the phrenic nerve and irritation of this portion causes pain in the neck. The peripheral rim of the diaphragmatic pleura is innervated by sensory fibers of the intracostal nerves and irritation of these areas give rise to pain in the lower part of the thorax, lumbar region and abdomen.

Báron [29] reported the case of a young man with a nonhemorrhagic bayonet wound in the ninth left intercostal space. The patient had severe paroxysms of coughing, and pain in the left shoulder, which were relieved by blocking the phrenic nerve by the injection of procaine hydrochloride.

Abnormalities in the course of the phrenic nerve are common. In 1880, Walsham [30] called attention to abnormalities in its course over the anterior scalene muscle and the nerve forming a loop around the subclavian vein by a branch from the nerve to the subclavius muscle. In recent years increased interest has been stimulated by the use of phrenicotomy in the treatment of disease. The early results were not always satisfactory and in many instances the cause of failure was attributed to continued function of the diaphragm after the nerve in the cervical region had been cut. This led to detailed studies of the anatomy and physiology of the phrenic nerve.

In 1922, Felix published a review of his experimental, anatomic and clinical observations following evulsion of the phrenic nerve. His

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26. Ramström (footnote 21).

27. Ferguson, John: The Phrenic Nerve, Brain **14**:282, 1891.

28. Capps, J. A.: An Experimental Study of the Pain Sense in the Pleural Membranes, Arch. Int. Med. **8**:717 (Dec.) 1911.

29. Báron, Alexander: Blockade des Nervus phrenicus bei Verletzung des Zwerchfells, Zentralbl. f. Chir. **50**:442, 1923.

30. Walsham, W. J.: Abnormal Course of the Phrenic Nerve, St. Barth. Hosp. Rep. **16**:100, 1880.

work explained why unsatisfactory results had followed simple phrenicotomy. He stated that in from 20 to 25 per cent of persons an accessory phrenic nerve is present, and that the failure of operation was due to accessory branches along the course of the nerve which ultimately restored the function of the diaphragm.

In 1920, Goetze recognized the presence of accessory branches and performed phrenicotomy and division of the nerve to the subclavius muscle. In 1925, he published a report of his anatomic investigations of thirteen cadavers, and found that in seventeen of twenty-five phrenic nerves he was able to show a branch of varying size from the subclavius (or nerve to the subclavius) going to the main stem of the phrenic nerve. He described the radical operation, in which he cut the subclavius nerve and removed from 7 to 8 cm. of the phrenic nerve, dissecting down to the small branches in the upper part of the thorax. He also described a method of temporal radical phrenicotomy in which the subclavius nerve is cut and the phrenic nerve frozen with ethyl chloride, thereby producing temporal paralysis.

Hellin claimed priority for having suggested the presence of accessory branches to the phrenic nerve and for having raised the first doubt as to the paralysis of the diaphragm and the therapeutic usefulness of simple phrenicotomy, stating that it was his work which led to further investigation of the structure and course of the phrenic nerve and which resulted in change in the operative procedure.

Plenk and Matson [31] have called attention to many variations in the anatomy of the phrenic nerve.

Schlaepfer [32] concluded, from his experiments, that the phrenic nerve was the only motor nerve to the diaphragm in dogs. He stated that Bertilli, Russel, Fuchs, Eisler, von Gössnitz and Ramström expressed the belief that the phrenic nerve is the only motor nerve supply to the diaphragm, and that Luschka, Gansina, Cavalié, Henle, and others, believed that the lower intercostal nerves send motor branches to the muscle, basing their conclusions on anatomic dissection.

PHYSIOLOGY

That paralysis of half of the diaphragm does not produce remarkable clinical or pathologic phenomena has been observed clinically and demonstrated by animal experimentation, but there is still some difference of opinion as to the physiologic effects.

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31. Plenk, A., and Matson, R. C.: Zur Phrenicotomiefrage, Beitr. z. Klin. d. Tuberk. **62**:350, 1925.

32. Schlaepfer, K.: The Phrenic as the Nerve of Motor Innervation of the Diaphragm, Bull. Johns Hopkins Hosp. **34**:195, 1923.

Before phrenicotomy was used in the treatment of disease, Oppenheim [33] stated that it is difficult to recognize unilateral phrenic paralysis as the functional disorder is very slight.

In 1890, Hare and Martin [34] performed experiments on rabbits, cutting one and both phrenic nerves to disprove the prevailing idea that this procedure is followed by death. They found that the diaphragm was reduced to a fibrinous membrane from atrophy after sixty days and that death followed only in young rabbits after section of both nerves under certain conditions.

Neuhöfer [35] reported the case of a boy, aged 8, on whom bilateral phrenicotomy had been performed, and three years later the diaphragm was found to be practically immobile. The boy had developed normally and the thoracic respirations had answered all practical purposes.

Yates [36] stated that abnormal spasmodic contractions of the diaphragm preceding flaccid relaxation and the forcing of the diaphragm into a high position occur in man and in animals when irritation of either visceral or parietal pleura is induced or develops spontaneously. He stated that besides the clinical observations indicating the efficacy of improving the quality and increasing the quantity of blood delivered to the lungs in helping patients to combat tuberculosis, other observations have shown that animals and man, whether suffering from acute or chronic pleuropulmonary disease and wounds, could be similarly benefited. He is convinced that pleuropulmonary resistance, defense and repair are at their best when the lung is in a position near mean inflation and its excursions above and below this mean are restricted. He believed this position is assured by paralysis of the diaphragm and is manifested in natural defense responses.

Lemon,[37] in a series of experiments on dogs, has shown that unilateral diaphragmatic paralysis does not change in any demonstrable way the movements or shape of the wall of the chest. The only effect on the thoracic cavity is to lessen the long diameter. The volume of the lung is decreased by lessening one dimension only. The intrapleural tension was not altered and the functional capacity of the animals seemed to be normal. He stated that phrenicotomy was attended with little

33. Oppenheim, Hermann, translated by Alexander Bruce: Text-Book of Nervous Diseases, London, S. C. Foulis, 1911, vol. 6, p. 423.

34. Hare, H. A., and Martin, Edward: The Effect of Section of the Phrenic Nerves, Lancet **1**:124 and 185, 1890.

35. Neuhöfer, Paul: Ueber die Bedeutung pathologischer und künstlicher Phrenicusschacligungen für die Einstellung und Funktion des Zwerchfells, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **35**:1, 1922.

36. Yates, J. L.: Rationale of Operations Helpful in Promoting Recoveries from Pulmonary Tuberculosis, Arch. Surg. **14**:369 (Jan.) 1927.

37. Lemon, W. S.: Physiologic Effect of Phrenic Neurectomy, Arch. Surg. **14**:345 (Jan.) 1927.

or no risk to the dogs; it did not cause impairment of function in general or of respiratory function in particular. The compensation was sufficient to overcome the loss in function of half of the diaphragm or indeed that of the whole diaphragm when both phrenic nerves were severed. The animal was competent to carry on its usual activities without embarrassment or dyspnea. Atrophy in the diaphragm appeared early, but paralysis appeared at once. The paralysis on the side of the section was complete and the atrophy uniform.

This paper is presented for the purpose of reporting the use of phrenic evulsion as a palliative measure in cases of diaphragmatic hernia, in which radical surgical procedures are contraindicated. I have not found in the literature any reference to its use as the only surgical treatment in cases of diaphragmatic hernia. I am also reporting the use of phrenicotomy and phrenic emphraxis as an aid to the radical operative closure of the opening by the abdominal approach. Two cases are discussed in which the paralyzed diaphragm was utilized in closing the operative defect after removal of large malignant tumors of the wall of the chest, with involvement of the diaphragm in one case.

In January, 1926, I operated on a patient with a large traumatic hernia of the foregoing type. There was loss of tissue and the margins of the opening could not be approximated. After the muscle was relaxed by phrenicotomy, the opening was easily closed. The result has been very satisfactory. I have heard from the patient recently. Symptoms have not recurred, and he is doing heavy work daily.

Permanent or temporary paralysis of the diaphragm is necessary in the repair of diaphragmatic hernia in only a selected group of cases. In the last year I have operated in ten cases of diaphragmatic hernia, employing phrenic evulsion in four cases, phrenicotomy in one case and phrenic emphraxis in one. The percentage of cases in this group is higher than usual because of the exceptional clinical and pathologic data as shown in the case histories. In reviewing the histories of patients seen at the clinic, suffering from chronic diaphragmatic hernia, it is interesting to note that many have had symptoms over a period of years. Their chief complaint is usually "stomach trouble" characterized by periodic severe "attacks," which in many instances have been diagnosed as gallbladder disease, angina pectoris, perforating ulcer and cardiospasm. The onset may or may not be associated with injury. The patients have more or less indefinite indigestion but get along fairly well if they are careful as to quality and particularly as to quantity of the food taken or do not exert themselves physically. The periodic attacks come on suddenly, usually after some dietary indiscretion or excessive physical exertion. Patients are seized with severe deep epigastric pain which often radiates to the back of the neck or shoulder.

In cases in which the stomach only is incorporated in the hernial opening, there is nausea and distention with difficulty in belching gas or in vomiting. There is a sense of fulness and oppression in the thorax associated with dyspnea and cardiac embarrassment from upward pressure on the lung and pericardium. The severity of symptoms depends on the amount of herniated abdominal viscera. If the bowel is incorporated in the hernia, the symptoms are usually more severe and are associated with signs of intestinal obstruction of varying degree. Cardiac stimulants and large doses of morphine are often given without relief. But in most instances they are instantly relieved by belching of gas or vomiting. After a number of attacks, patients promptly resort to this treatment and in some instances learn to wash their stomach with the aid of a tube. The stomach may be in the thorax only during the attacks, and after relief is obtained it drops back into its normal position below the diaphragm. This is exemplified in one patient of this group. In most instances the stomach is constantly in the thoracic cavity, but the patients have comparatively little trouble as long as they eat moderately and maintain good elimination of the intestinal tract.

In most instances it is the severe periodic exacerbation of symptoms that forces the patient to seek relief. A rational hypothesis for these periodic attacks is that increased intra-abdominal pressure primarily forces the stomach through the opening in the diaphragm into the thorax, as is the case in any abdominal hernia. After the stomach has passed through the diaphragm there are several factors which tend to drag more of the abdominal viscera into the thorax: the peristaltic action of the stomach, the suction of the negative pressure of the thorax and, probably most important, the pumping action of the diaphragm. The increased intra-abdominal pressure not only forces the stomach through the opening but also prevents its return. In a short time this large incarcerated portion of the distended stomach interferes with the function of the diaphragm which with irritation causes severe spasmodic contraction of the muscle in its effort to counteract this abnormal condition. The spasm of the muscle and the irritation of the diaphragmatic pleura produce the reflex pain in the shoulder and often cause reflex cardiospasm which acts as a vicious circle by preventing release of the intravisceral pressure by vomiting. The contraction of the muscle on the stomach often produces an hour-glass deformity and prevents the emptying of that portion above the diaphragm. It increases the pressure in the thorax and prevents the release of the intravisceral pressure of this segment of the stomach. This intravisceral pressure tends to increase the amount of herniated stomach and often is difficult to overcome even with a stomach tube which will only relieve the portion of

the stomach below the diaphragm. The deformity of the cardia and the lower part of the esophagus caused by the abnormal position and pressure causes difficulty in belching gas and in vomiting in cases in which cardiospasm is not present. In this way the action of the diaphragm is not only a large factor in the production of the hernia, but it also maintains the herniated contents in the thorax. The increased intra-abdominal pressure causes cardiac embarrassment and dyspnea.

Based on this hypothesis, the action of the diaphragm is an important factor in the production of these attacks of incarceration. Paralyzing the muscle by phrenicotomy prevents the pumping action of the diaphragm on the herniated viscera and the relaxed muscle cannot be thrown into spasmodic contraction. The nerve communications are destroyed, which prevents reflex cardiospasm and pain in the shoulder. In those cases in which the stomach is permanently in the thorax, the stomach will move synchronous with the diaphragm, and when there is a paradoxic motion this might tend to lessen the amount of herniated contents. For these reasons I believe that relaxation of the diaphragm by phrenic neurectomy is of value as a palliative measure in cases of diaphragmatic hernia in which radical closure of the opening in the diaphragm is contraindicated. In case of multiple complaints and in case it is difficult to evaluate the amount of symptoms due to the hernia, it is best first to perform phrenic emphraxis by crushing or injecting the nerve as a test before completely destroying the nerve supply to the diaphragm.

I have instituted treatment in three such cases in the last year with good results. In two of the cases more than six months have elapsed which, I believe, is sufficient time in which to determine the efficiency of the treatment. In the third case about a month has elapsed and the result is satisfactory at the present time, but it is too soon to consider the result conclusive. The summaries of the histories of these cases show why radical operation was contraindicated in each case; they also exemplify the type of case in which I believe this procedure is indicated.

REPORT OF CASES

CASE 1.—A woman, aged 50, was admitted to the Mayo Clinic on Oct. 7, 1927, complaining chiefly of cardiac and gastro-intestinal symptoms. The gall-bladder had been removed nine years previously, and except for an occasional "bilious attack" consisting of nausea and vomiting of bile, she was well until two years before examination. The bilious attacks had become progressively more frequent and severe during the last two years and now occurred every few days. She was nauseated most of the time, with marked epigastric bloating and difficulty in belching gas. Typical attacks started with bloating, then a sensation of tightness appeared across the upper part of the abdomen, and a cutting sensation across the back on swallowing. Pyrosis and pain in the back of the throat appeared three fourths of an hour after meals. These attacks

lasted from three to four days, then she obtained relief by repeated attacks of vomiting of food and bile, and gradual disappearance of the distention. For the last two and a half years, she had attacks of precardial pain associated with a sensation of constriction through the epigastrium; she was very dyspneic and had to be propped up on pillows. The attacks usually followed bilious attacks. There was edema of the ankles, which at times was extreme, and there were frequent slight attacks of palpitation of the heart, and dyspnea on exertion. She had lost 20 pounds (9 Kg.).

The systolic blood pressure was 150 and the diastolic 100. The pulse rate was 104 and regular. The electrocardiogram showed a rate of 102, sinus

Fig. 1.—On admission; diaphragmatic hernia, with about one fifth of the stomach protruding through the esophageal opening into the left side of the thorax.

tachycardia, incomplete bundle branch block, left ventricular preponderance, notches in P wave in derivation III and diphasic T wave in derivation I; the QRS interval was from 0.12 to 0.14 seconds. Gastric analysis revealed total acidity, 38, free hydrochloric acid, 24, and quantity, 80 cc. Roentgen examination of the stomach showed hernia of the cardia through the esophageal opening in the diaphragm (fig. 1).

A diagnosis was made of nontraumatic esophageal diaphragmatic hernia through the esophageal opening. It was believed that the attacks were due to

two distinct conditions: myocardial disease and incarceration of the stomach in the hernial sac. As the symptoms were severe and the hernia was such an important factor in the complaint, surgical intervention was necessary. Because of the severe cardiac injury, radical closure of the opening of the hernia would entail great operative risk and for this reason I thought it advisable to try first left phrenic neurectomy in the hope that paralyzing the diaphragm, thus preventing incarceration of the stomach in the hernial opening, would relieve the attacks so that radical operation would not be necessary.

On Oct. 26, 1927, left phrenic evulsion was performed under local anesthesia with removal of 11 cm. of the nerve. Convalescence was uneventful (fig. 2).

Fig. 2.—Nineteen days after left phrenic neurectomy; fixation of the diaphragm without protrusion of the stomach above; relief of gastric symptoms.

In a recent letter the patient said that she had been much relieved by operation, but had had several cardiac attacks which were not associated with the severe so-called bilious attacks and bloating with epigastric distress as before. There seems to be 50 per cent improvement in her condition. Most of her present trouble is due to her heart. The residual trouble which may be caused by the diaphragmatic hernia is not sufficient to require radical surgical procedures.

Case 2.—A woman, aged 63, was admitted to the clinic on Aug. 12, 1927, because of stomach trouble. Since childhood she had complained of intermittent regurgitation of food immediately or shortly after meals. About eight years

previously this regurgitation had become more difficult and deep epigastric distress appeared immediately after meals. This had become progressively more severe, particularly in the last two years. The distress was localized deep in the epigastrium and lower part of the thorax, often accompanied by pain in the back of the left side of the neck. There was a sensation as though food and gas would not pass a certain point deep in the epigastrium. These attacks of extreme distress appeared almost daily. All food seemed to disagree. She felt full and bloated with upward pressure causing palpitation of the heart and difficulty in breathing. She had been on virtually a starvation diet because of this distress which was relieved only by induced vomiting or gastric lavage with a stomach

Fig. 3.—On admission; diaphragmatic hernia with about four fifths of the stomach through the esophageal opening and extending to the right of the median line.

tube. There was a history of trauma of the upper part of the abdomen thirty years previously, but it is questionable whether this was a factor in the present complaint. There had been attacks of severe lumbar pain with radiation to the region of the bladder and some frequency. The urine had contained red blood cells and at one time gross hemorrhage had occurred from the bladder. Two months prior to examination severe pain had occurred along the right costal margin with radiation to the right shoulder requiring a hypodermic injection of morphine for relief. The patient was jaundiced the following day, and this

continued for about two weeks with residual soreness in the right costal margin for several days. She had several of these attacks, the first about nine years previously, but jaundice was never associated and attacks were entirely different from those due to the stomach complaint.

Examination showed moderate emaciation with slight icterus. Urinalysis revealed red blood cells and many pus cells. Renal function was 20 per cent. Cystoscopic examination disclosed a large stone occluding the right ureter. There was complete uterine prolapse with cystocele. An outline of the gallbladder was not visible in the roentgenogram because of cholecystic disease. Roentgenograms of the stomach revealed diaphragmatic hernia; three fourths of the stomach was

Fig. 4.—Left nontraumatic diaphragmatic hernia extending into the right side of the thorax; left phrenic neurectomy; complete relief of gastric symptoms.

through the esophageal opening extending into the left side of the thorax and pushing over into the right (fig. 3). A diagnosis was made of nontraumatic left diaphragmatic hernia, a stone completely blocking the right ureter, chronic cholecystitis with cholelithiasis and complete uterine prolapse.

The jaundice entirely cleared after the first week in the hospital. Because of the low renal function it was thought advisable to remove the stone from the right ureter first, which was done on Sept. 29, 1927. The stone measured 2 by 1 cm.

Convalescence was essentially uneventful but there was little improvement in renal function since the right kidney was practically functionless from the long-

standing disease. The diaphragmatic hernia was considered of next importance and because of the patient's poor general condition and low renal function, it did not seem advisable to subject her to a serious operation, as would be entailed in the radical procedure of closing the hernial opening; therefore I decided on left phrenic evulsion in the hope that by relieving the spasm of the diaphragm, thereby preventing incarceration of the stomach in the hernial opening, her symptoms would be alleviated sufficiently to defer the radical closure of the opening to a time when her general condition would permit a more extensive operation. On October 26, left phrenic evulsion was performed with removal of about 24 cm. of the phrenic nerve (fig. 4).

Fig. 5.—On admission; herniation of the cardiac end of the stomach through the esophageal opening.

Convalescence was uneventful. The patient was dismissed from observation on November 29. In a recent letter she stated that her general condition is very good. She is eating regular meals for the first time in four years without distress and gastric symptoms have disappeared.

CASE 3.—A man, aged 63, was admitted to the clinic on March 1, 1928. He had had stomach trouble for three and a half years. During the last three years, the pain appeared in the epigastrium soon after meals, becoming more or less constant and gradually more severe; it was not relieved by the use of soda or the ingestion of food, but usually by vomiting which was accomplished with

considerable difficulty. For two months he vomited after almost every meal; the vomiting was associated with severe epigastric distress. He was often awakened at night with epigastric pain which was relieved by belching of gas or vomiting. For the last two months severe pain had been present to the right of the sternum. The patient ate but little because of the pain; he had lost 16 pounds (7.3 Kg.) during the past two years.

Examination revealed moderate myocardial degeneration and arteriosclerosis; a systolic blood pressure of 160 and a diastolic of 100; benign hypertrophy of the prostate; a few pus cells and red blood cells in the urine; total gastric acidity 16 and no free hydrochloric acid in 200 cc. Roentgenograms of the stomach

Fig. 6.—Eight days after left phrenic evulsion; elevation of the left side of the diaphragm. Practically none of the stomach is seen above the diaphragm; complete relief of symptoms.

showed one fifth of the cardiac end in the left thoracic cavity through the esophageal opening of the diaphragm (fig. 5).

A diagnosis was made of nontraumatic left diaphragmatic esophageal hernia. Because of the patient's age, and because of the myocardial degeneration associated with arteriosclerosis and hypertension, radical operation was contraindicated and left phrenic neurectomy was advised.

On March 14, 1928, left phrenic evulsion with removal of 18 cm. of the phrenic nerve was performed under local anesthesia. Convalescence was unevent-

ful and the patient was dismissed at the end of two weeks (fig. 6). He was completely relieved of the pain along the border of the sternum from the time of operation. His appetite has improved and he has practically no distress after meals. Although there has been complete relief of symptoms, it is entirely too soon to evaluate the permanency of results.

I prefer the abdominal approach in the repair of diaphragmatic hernia. Phrenicotomy is valuable in cases in which the hernial openings are large and the margins cannot be approximated because of the con-

Fig. 7.—Diaphragmatic hernia with more than half of the stomach in the left pleural cavity; large opening in the left dome of the diaphragm.

traction of the muscle of the diaphragm, or if approximation is obtained there is undue tension on the sutures. The relaxation of the diaphragm will often permit complete closure of the opening without tension, which is essential if good healing is to be obtained. In some cases the opening cannot be closed even by relaxing the diaphragmatic muscle. There is usually an enormous opening with marked loss of muscle tissue or the muscle may be partially torn from the wall of the chest. These openings may be closed by narrowing the diameter of the

thorax by posterior extrapleural thoracoplasty of the lower ribs associated with phrenicotomy. This is a procedure of great magnitude, but as patients who require this type of treatment are usually in extremus, the risk of such an operation is justified. If patients are extremely obese and the opening in the diaphragm is difficult to expose, temporary relaxation of the muscle by phrenic emphraxis is of great aid in the technical closure of the opening. The relaxed muscle and the limited mobility of the diaphragm permit a more accurate approximation of the margins of the opening. The exposure of the diaphragm is greatly facilitated and I believe that the healing of the repaired muscle is aided. These indications are exemplified in cases 4, 5 and 6.

Fig. 8.—Left traumatic diaphragmatic hernia; incision of cervical phrenic neurectomy and also incision of abdominal approach through old postoperative hernia from a previous operation.

CASE 4.—A man, aged 48, was admitted to the clinic on Oct. 31, 1927, with pain in the epigastrium and the left shoulder of four weeks' duration. One month previously he had been crushed between a steering wheel and the seat of a truck, and was unconscious for about two hours. The first time he took any quantity of food, which was on the third day after the accident, he was seized with severe epigastric pain which quickly moved to the left shoulder, corresponding to the free border of the trapezius muscle about 7.5 cm. from the point of the shoulder and extending to the occiput. The pain was associated with dyspnea and palpitation of the heart. Amyl nitrate was given without relief and then ½ grain (32 mg.) of morphine, hypodermically, was given, but did not control the pain. The pain continued for one and a half hours, at which time more amyl nitrate was used shortly after which he vomited, with instant relief

of pain. He had six or seven of the attacks of pain in the last month, the pain being most severe in the area of the shoulder. Between attacks he was practically free from symptoms. He could eat small amounts of food without any discomfort but a large meal would bring on an attack. Roentgenograms elsewhere revealed a rupture of the diaphragm with the stomach in the left thoracic cavity.

At the time of admission, the patient had had an acute cold for several days. A roentgenogram was taken and showed the stomach in the normal position below the diaphragm; the diaphragm moved normally. Because of the acute

Fig. 9.—Two months after phrenic evulsion and abdominal repair of the hernial opening in the dome of the diaphragm showing the entire stomach below the diaphragm which is moderately elevated.

cold it was not thought advisable to operate at this time. Three days later he had a typical acute attack which was relieved by vomiting, and a roentgenogram taken the following day, after complete cessation of pain, showed the stomach again in normal position. He was advised to wait until he was entirely over the acute cold and then return for operation.

The second examination revealed a large postoperative epigastric hernia from a previous operation. A roentgenogram revealed a shadow in the left thoracic cavity, probably a shadow of the stomach. A diagnosis of traumatic left

diaphragmatic hernia, with two thirds of the stomach in the left thoracic cavity, was made (fig. 7).

Because of the large size of the opening, which appeared from the roentgenogram to be in the dome of the diaphragm, preliminary phrenic evulsion was decided on and performed on December 7. Three days later the opening in the diaphragm was closed by an abdominal approach, under intratracheal anesthesia. The large opening in the left anterior dome of the diaphragm measured about 10 cm. in diameter. About two thirds of the cardiac end of the stomach, the spleen and a small portion of the large intestine were in the left thoracic cavity. The hernial opening was closed with interrupted silk

Fig. 10.—On admission; transverse colon, splenic flexure and descending colon in the left side of the thorax extending to the apex of the lung.

mattress sutures (fig. 8). Convalescence was uneventful. There was moderate collapse of the left lung following the operation, and slight dyspnea. Air was aspirated from the left thoracic cavity to hasten reexpansion of the lung.

The patient returned for observation on Feb. 3, 1928. Roentgenograms of the chest and stomach showed moderate elevation of the diaphragm and the stomach entirely below the diaphragm (fig. 9). He was completely relieved of gastric symptoms.

This case demonstrates very well the syndrome so commonly noticed in patients suffering from diaphragmatic hernia. The attacks are probably due to

the increased intra-abdominal pressure forcing the stomach through the opening in the diaphragm, after which the pumping action of the diaphragm, together with the negative pressure in the thorax, gradually draws more of the stomach into the thorax until incarceration of the stomach occurs with spasmodic contraction of the diaphragm causing dyspnea with palpitation due to cardiac embarrassment, and in many instances reflex cardiospasm. At operation, the complete closure of this large opening was possible because of the relaxation and immobility of the muscle resulting from phrenic evulsion causing paralysis of the diaphragm.

CASE 5.—A man, aged 47, was admitted to the clinic on Nov. 7, 1927, complaining chiefly of dyspnea, inability to eat and weakness. Five weeks previously he had been hurled from his car in an automobile accident, sustaining multiple lacerations of the face, neck and body and fracture of the right wrist and left

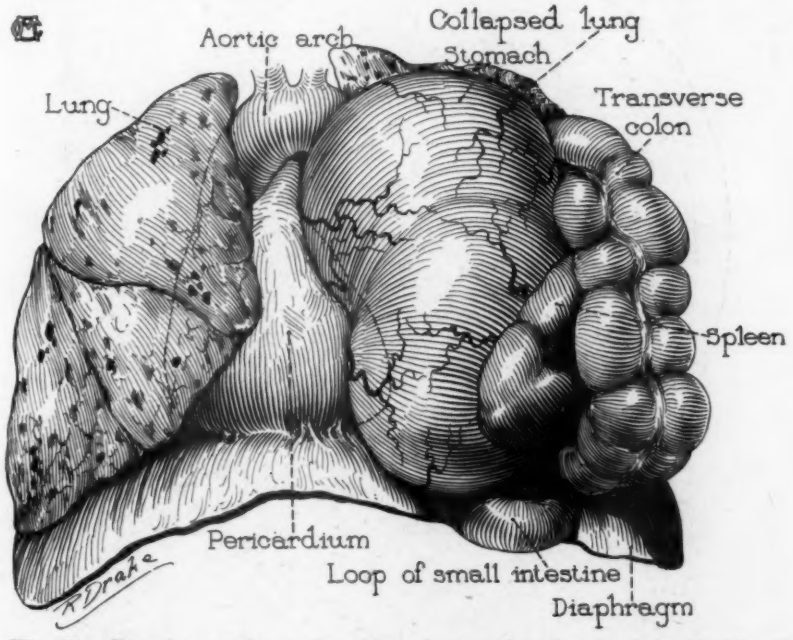

Fig. 11.—Drawing made at the time of operation for large traumatic diaphragmatic hernia; the entire stomach, half of the transverse colon, the splenic flexure and the descending colon, many loops of the small intestine and the spleen are seen in the left side of the thorax with complete collapse of the left lung, and partial collapse of the right lung.

lower ribs. He had vomited a great deal the first twenty-four hours, but the vomitus did not contain blood. Blood-streaked mucus was expectorated for several days. Cough and marked dyspnea were present. There was much pain and tenderness over the lower left side of the chest, the left shoulder, and pain in the upper part of the abdomen where he had been struck by the steering wheel. After several days, chills and fever developed; these gradually subsided after five or six days. Much of the pain had subsided, but the dyspnea was marked even at rest and was aggravated when the patient was lying down. He had difficulty in swallowing food, particularly solid food, and discomfort in the upper left side of the chest after eating. He had lost 30 pounds

(13.6 Kg.) since the accident. The pain and dyspnea prevented him from lying down, and he obtained little relief from sedatives. He had had nephritis and cardiac trouble for years.

The patient appeared very ill. The lacerations of the scalp, face and legs had healed. Breath sounds were absent over the entire lower part of the chest below the level of the third rib. The heart was displaced to the right. A roentgenogram showed a large shadow in the left side at the thorax, probably of abdominal viscera through the rupture in the diaphragm, also complete collapse of the left lung with displacement of the mediastinum to the right and partial collapse of the right lung. The transverse colon,

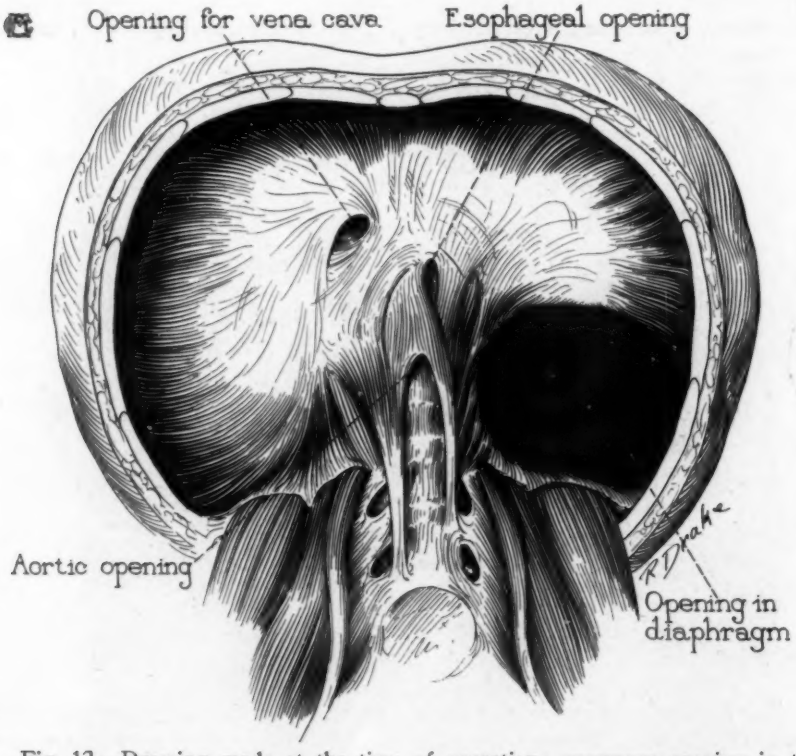

Fig. 12.—Drawing made at the time of operation; enormous opening in the left side of the diaphragm which has been torn from the wall of the chest.

splenic flexure, descending colon and loops of small intestine were shown in the left side of the thorax extending to the apex (fig. 10). Fluoroscopic examination failed to disclose any function of the diaphragm on the left side except close to the median line. A diagnosis was made of traumatic left diaphragmatic hernia with the diaphragm torn from the wall of the chest and multiple abdominal viscera in the left thoracic cavity. Because of the severity of the symptoms, operation was advised. The enormous opening in the diaphragm, which had been torn from the wall of the chest, could not be repaired without first paralyzing the diaphragm by phrenic evulsion and dropping the wall in by posterior thoracoplasty so that the diaphragm could be resutured to it (figs. 11 and 12).

On November 9, left phrenic evulsion was performed, and two days later lower posterior extrapleural thoracoplasty, the posterior segments from the seventh to the eleventh ribs, inclusive, being removed. Four days later the diaphragm was repaired through an abdominal approach; an intratracheal ethylene anesthetic was used, the preliminary administration being carried out with the patient in the upright position because of extreme dyspnea.

As practically the entire diaphragm was torn from the wall of the chest, the opening was enormous and extended posteriorly to the crus, leaving a large oval-shaped rent about 12.5 cm. in diameter. The entire stomach, spleen, half of the transverse colon, splenic flexure and a portion of the descending

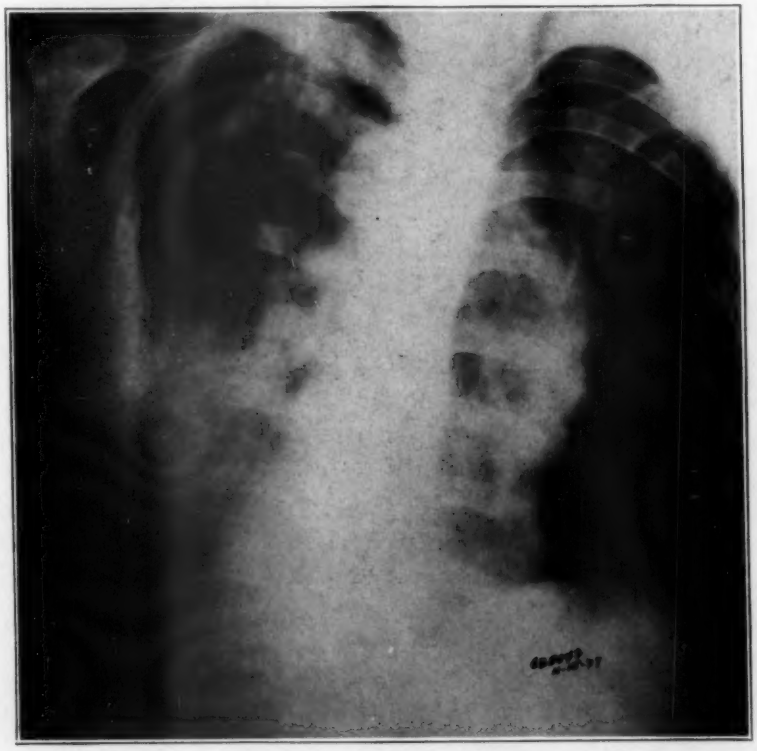

Fig. 13.—Seven days after left phrenic neurectomy, four days after posterior thoracoplasty, removing posterior segments of the lower seven ribs, and one day after repair of the opening in the diaphragm through an abdominal approach; complete reexpansion of the right lung with partial reexpansion of the left lung is shown. All abdominal contents were removed from the left side of the chest.

colon with many loops of small intestine were in the left side of the thoracic cavity. The abdominal viscera were replaced in the abdomen and the relaxed diaphragm was resutured to the wall of the chest and to the small fringe of torn diaphragm remaining along the costal attachment. There was some difficulty in closing this large opening with the torn diaphragm, but owing to the relaxed muscle and shortened diameter of the chest, a satisfactory closure was made

and the torn margin of the diaphragm was sutured to the wall of the chest with interrupted linen sutures. At completion of the operation the patient's condition was as satisfactory as could be expected. Respirations were 30 and not labored as before operation, and the pulse rate was 130. Transfusion was given immediately after operation, which greatly improved the volume of the pulse. Convalescence was uneventful for the first twenty-four hours (fig. 13). The second day, respirations increased to 40. The patient was immediately transferred to the oxygen chamber and his condition showed some improvement for the first twelve hours; then the urinary output decreased and

Fig. 14.—On admission; nontraumatic esophageal diaphragmatic hernia with three fourths of the stomach above the diaphragm in the left side of the thorax and extending posteriorly into the right side of the thorax.

the pulse rate increased. The third day he became comatose with increase in respirations, and he died on the fourth day.

Necropsy showed a small area of bronchopneumonia in the right lung with nephritis and moderate myocarditis. The site of operation showed a very satisfactory closure of the opening in the diaphragm.

There were many factors in this patient's condition to make an operation of this magnitude hazardous even though done in stages, but the chief reason he did not survive the operation was because of the

recent lowered resistance added to his already poor general condition at the time of the accident. The lowered resistance was due to the loss of weight, almost 1 pound a day, since the accident, the constant exertion in order to breathe, and the pain and inability to obtain any rest even by the use of sedatives. Dextrose and sodium chloride solution were given intravenously during the course of the operations. At necropsy, it was impossible to attribute death to the lesions found in the right lung, kidney and heart, as these organs were only moderately

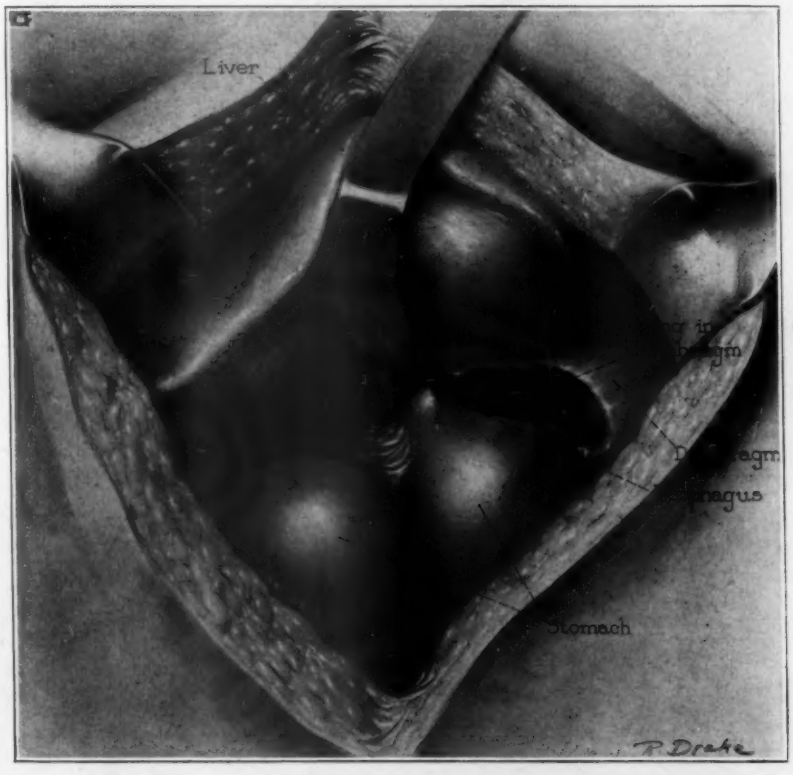

Fig. 15.—Drawing at time of operation after removal of the stomach from the thoracic cavity. A moderate sized diaphragmatic hernial opening is shown just to the left and continuous with the esophageal opening.

involved and it was decided that the lesions were only contributory factors.

Case 6.—A woman, aged 45, was admitted to the clinic on Aug. 22, 1927, complaining of attacks of abdominal colic of four or five years' duration. The pain in the epigastrium had been severe, radiating around both sides (more on the left) to the back. Attacks came on suddenly at irregular intervals, were always brought on by taking food and were associated with bloating and belching

of gas. Vomiting was difficult. Morphine was required for relief. The pain was more severe when the bowels were constipated. Attacks usually lasted about two hours unless morphine was taken; they might occur every day for a week and be absent as long as a month if care in the diet was exercised. Chills, fever or jaundice had not been present. Dyspnea was present on exertion and accompanying attacks. Precardial pain was always secondary to pain in the epigastrium. The condition had been diagnosed gallstones and angina pectoris.

The general examination was essentially negative except for moderate enlargement of the heart with accentuation of aortic second sounds. The patient weighed 205 pounds (93 Kg.) and was 5 feet 5 inches (165.1 cm.) tall. The

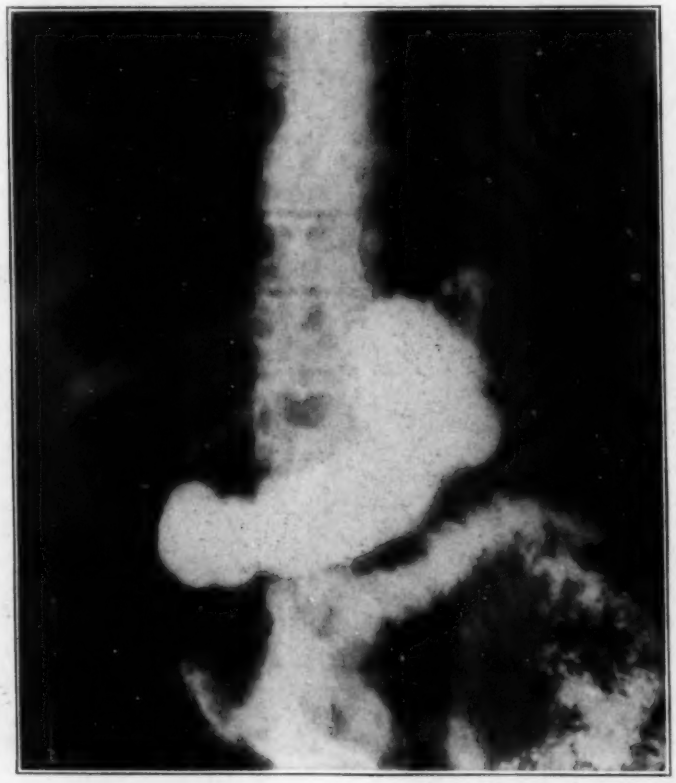

Fig. 16.—Two and a half weeks after preliminary phrenic emphraxis and abdominal closure of the hernia; the entire stomach is below the diaphragm; fluoroscopic examination showed the moderate motion and elevation of the left side of the diaphragm.

urinalysis was essentially negative. The hemoglobin was 64 per cent; erythrocytes numbered 3,920,000 and leukocytes 7,100. The Wassermann reaction of the blood was negative. Roentgenograms of the thorax showed moderate enlargement of the heart. Roentgenograms of the gallbladder, kidneys, ureter and bladder were negative. A diaphragmatic hernia with about three fourths of the stomach through the esophageal opening was shown (fig. 14). A diagnosis of nontraumatic left diaphragmatic hernia was made.

The operative risk was increased because of the obesity. On August 30, preliminary phrenic emphraxis was accomplished by injecting the left phrenic nerve, and the hernia was repaired through an abdominal approach. About three fourths of the cardiac end of the stomach was in the left side of the thorax and extended posteriorly into the right side (fig. 15). The opening in the diaphragm was from about 5 to 7.5 cm. in diameter just to the left of the esophagus and was closed with interrupted linen sutures. The exposure and closure of the opening was difficult but was greatly facilitated by the immobile and relaxed muscle, and it was possible to overlap the muscle about 1.75 cm. in the closure which I believe would not have been possible in a normally con-

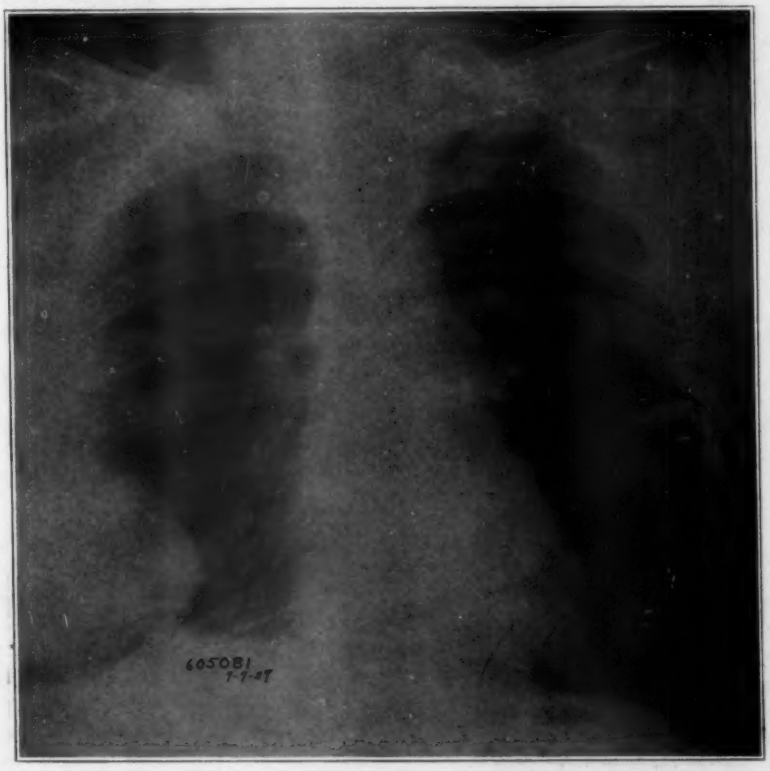

Fig. 17.—Roentgenogram of the thorax on admission; tumor (endothelioma) of the right wall of the chest involving the sixth, seventh, eighth and ninth ribs; large intrathoracic projection.

tracting diaphragm. The temporary paralysis of the muscle will aid the healing process of the approximated margins of the hernial opening and increase the possibility of firm union with cure of the hernia.

Convalescence was uneventful. A roentgenogram taken fourteen days after the operation showed the stomach to be in a normal position (fig. 16). Fluoroscopic examination showed restricted normal motion of the left side of the diaphragm. The patient was dismissed from the hospital on the fifteenth day and from observation on the twentieth day after operation. Fluoroscopic exam-

ination showed increasing function of the left side of the diaphragm. A recent letter stated that symptoms had not recurred.

Relaxation of the diaphragm by phrenicotomy may also be of value in the treatment of tumors low on the wall of the chest and of tumors of the wall of the chest involving the diaphragm. In these cases, the relaxed muscle is used to maintain a closed thorax by repairing the defect remaining after removal of the diseased tissue. I have utilized

Fig. 18.—Drawing made at the time of operation; removal of the entire involved wall of the chest including the sixth, seventh, eighth and ninth ribs. The large intrathoracic projection of the tumor is shown.

this procedure in two cases in the last year. In both cases malignant tumor was removed which required wide excision of the entire wall of the chest, and in one case partial excision of the diaphragm. In both instances, the phrenic nerve was cut at the time of the removal of the tumor and in one it was cut through a separate incision in the neck. In the other case the phrenic nerve was cut transpleurally before the

portion of the diaphragm involved was resected. In both cases the thorax was closed without drainage and the convalescence was uncomplicated, with the exception of pleural effusion requiring two or three aspirations. The lung remained expanded in both cases. A brief summary of these cases follows.

CASE 7.—A man, aged 37, was admitted to the clinic on July 6, 1927, because of tumor of the right side of the chest. He had always been well until ten months previously when he had noted a dull, dragging pain in the lower right

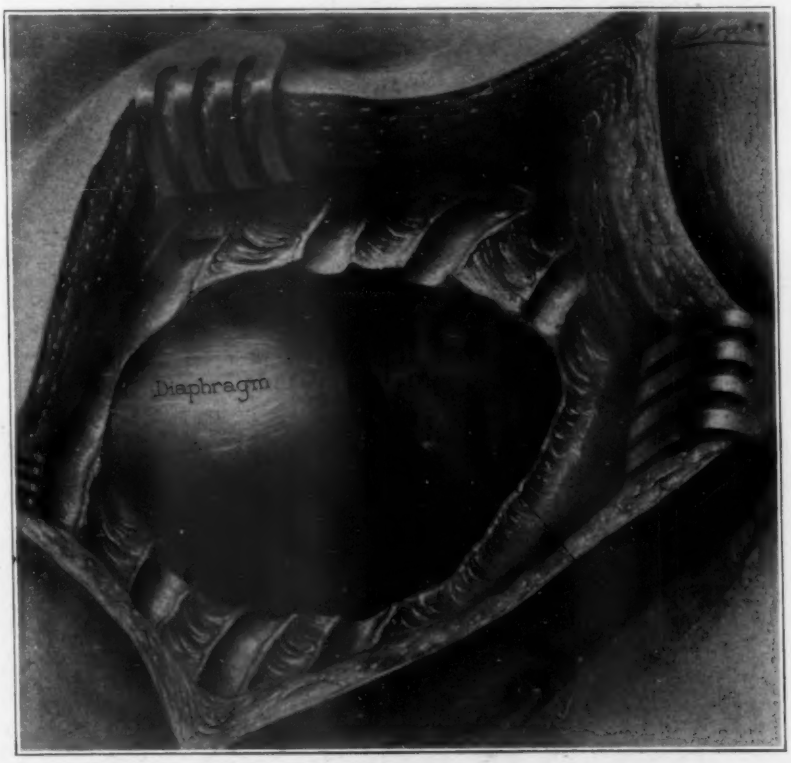

Fig. 19.—Enormous defect in the right side of the wall of the chest after complete removal of the tumor which involved the four ribs and the underlying pleura. The diaphragm and lower and middle lobes of the right lung are presented.

side of the chest; it was almost constantly present, aggravated by deep inspiration, and was increasing in severity. The pain was not preceded by any type of illness, infection or trauma. He had been treated for pleurisy; most relief had been gained by strapping the wall of the chest. Hot water bottles and rest also afforded relief. Three weeks prior to admission, he noticed a large diffuse swelling in the right side of the chest about the level of the nipple. Dyspnea and pain were prominent features of his complaint. He had lost 25 pounds (11.3 Kg.) during the past six months.

Examination revealed a nonpulsating, ovoid fixed tumor on the right side of the chest. The tumor was at the level of the sixth rib, anterior to the axillary line, and extending posteriorly into the axilla. There was no enlargement of the regional lymph glands and breath sounds were absent over this area. Urinalysis was negative. The hemoglobin was 75 per cent, the erythrocytes numbered 4,920,000, and the leukocytes 1,800. The Wassermann reaction of the blood was negative. Roentgenograms of the chest showed a circumscribed area of thickened pleura on the anterior lower right side of the wall of the chest, and an intrathoracic tumor with adhesions to the diaphragm (fig. 17). A clinical diagnosis was made of intrathoracic tumor (sarcoma) of the lower right side of the chest.

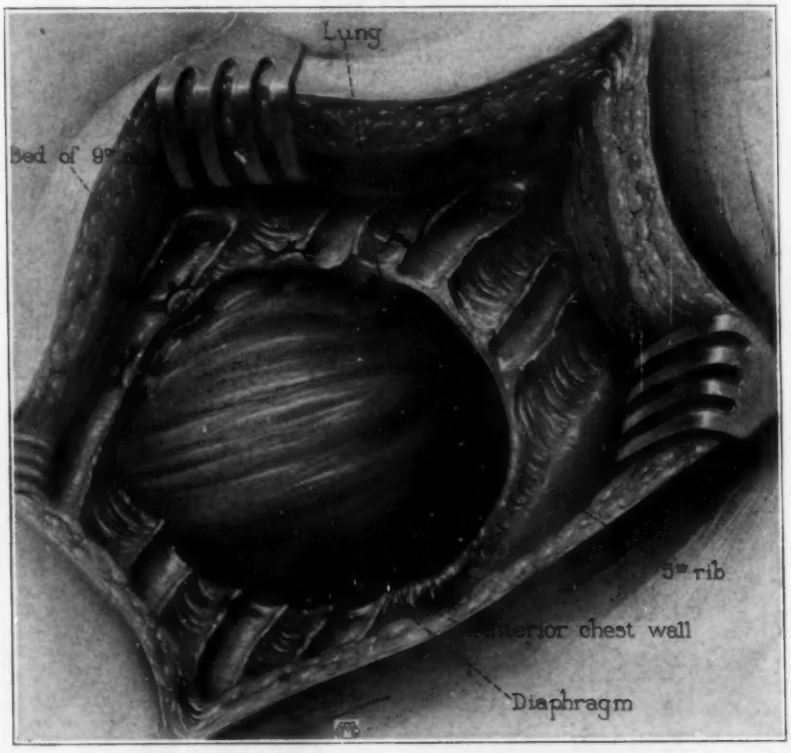

Fig. 20.—Closing the large defect in the right side of the wall of the chest with relaxed diaphragm after performing a right cervical phrenicotomy.

On July 12, operation was performed under intratracheal ethylene anesthesia. It was found that the tumor had infiltrated through the seventh and eighth intercostal spaces. A portion of the sixth rib was removed, the thoracic cavity was opened, and the intrathoracic portion of the tumor explored. It was adherent to the lung and involved the wall of the chest to within about 2.5 cm. of the attachment to the diaphragm, extended into the thorax about 8 cm., and was uniform in width. It was necessary to remove a portion of the sixth, seventh, eighth and ninth ribs with the entire wall of the chest to remove the involved tissue (fig. 18). This left a large opening, with the diaphragm presenting beneath the lower two thirds of the opening and lower lobe of the lung beneath

Fig. 21.—On dismissal five weeks after operation; the wound is healed; the patient's general condition is good.

Fig. 22.—On admission; large recurring tumor (endothelioma) in the lower left part of the thorax and lumbar regions; marked loss in weight.

the upper third (fig. 19). Right phrenicotomy was then performed to paralyze the right half of the diaphragm, which was utilized to close the opening in the wall of the chest by suturing it to the margins of the opening from the lower margin upward (fig. 20). The thorax was closed without drainage. The pathologist reported a highly malignant endothelioma. The tumor weighed 374 Gm.

Convalescence was uneventful except for a slightly bloody pleural effusion for which aspiration was carried out on three occasions. Following the operation the patient was given a course of roentgen treatment and was dismissed on August 24. The wound was entirely healed and the general condition was good (fig. 21). The dyspnea and pain in the lower right side of the chest had disappeared.

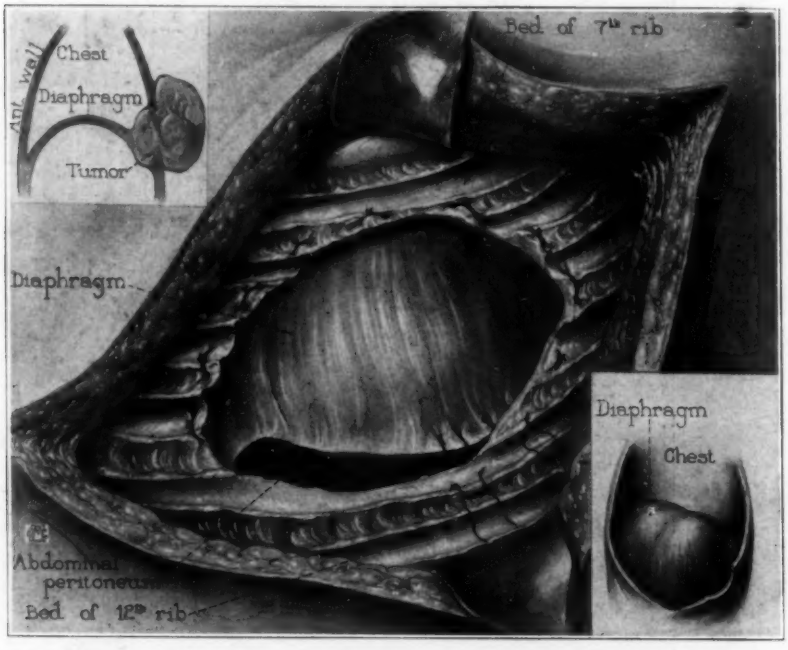

Fig. 23.—Drawing made at time of operation; resection from the seventh to the twelfth ribs inclusive with complete resection of the involved wall of the chest and diaphragm; the left thoracic cavity completely closed and separated from the abdominal cavity by repairing the defect in the wall of the chest with the relaxed diaphragm after intrathoracic left phrenicotomy. The insert at the top shows the location of tumor; the insert below, the resected portion of the diaphragm.

Three weeks after returning home, the patient began to have pain in the spine which radiated to the right hip. The pain gradually became more severe and in a short time dyspnea and blood-streaked sputum appeared. Death occurred in about four months, apparently from extensive metastasis.

Case 8.—A man, aged 28, was admitted to the clinic on Sept. 16, 1927, complaining of loss of weight, dyspnea, pain in the left shoulder and return of a tumor in the lower left portion of the wall of the chest. The patient's health

had been good until three years previously. While wrestling, two ribs had been fractured in the lower left side. He returned to work in three days. About nine months after the accident, he noticed a small fixed mass in the wall of the chest at site of the previous injury. The only symptom was slight pain on exertion. The growth progressed gradually; nine months later it was about 15 cm. in diameter, and operation was performed. The patient felt well for about eight months, then noticed recurrence of the tumor at the operative site. Growth had been very rapid and the tumor was then about 25 cm. in diameter.

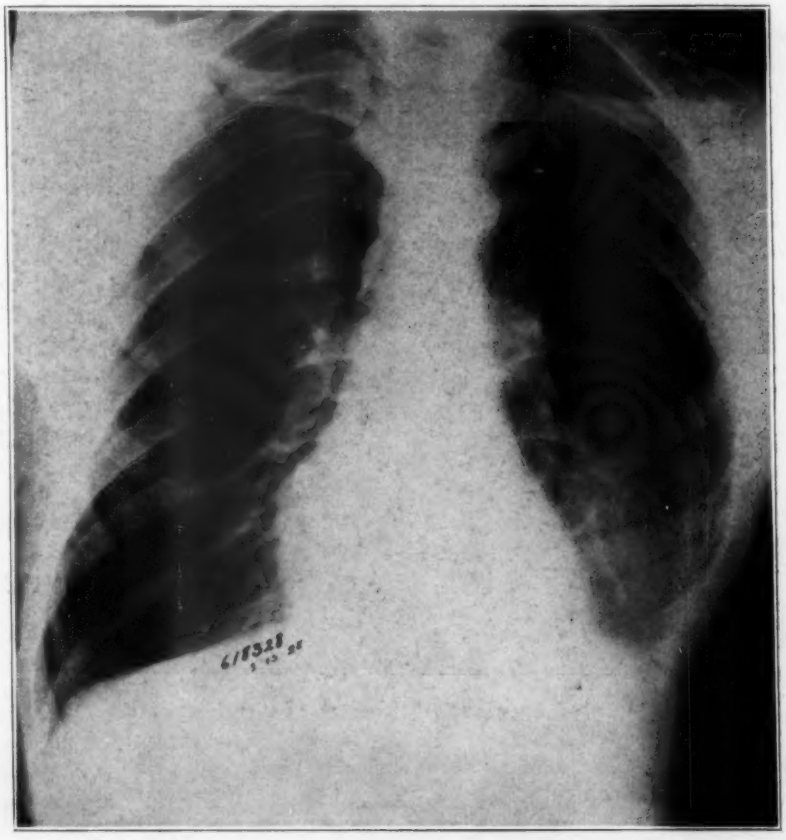

Fig. 24.—Six months after operation; resection from the seventh to the twelfth ribs inclusive with the involved wall of the chest and diaphragm. The heart is pushed slightly to the right, and there is no evidence of recurring tumor.

Weight had decreased from 145 to 104 pounds (65.8 to 47.2 Kg.). The patient was markedly dyspneic on exertion. He complained of attacks of pain in the left shoulder. About four months previously a plaster had been applied, and sloughing of the skin and an ulcerating discharge from the wound had continued since. One month previously, a severe hemorrhage had occurred from this sloughing wound. The patient had been shot in the back in the lower right portion of the thorax about two years before admission.

General examination disclosed an enormous tumor in the lower left portion of the thorax and lumbar region; the patient was greatly emaciated (fig. 22). The hemoglobin was 70 per cent, erythrocytes numbered 4,650,000 and leukocytes 16,100. The Wassermann reaction of the blood was negative. Roentgen examination revealed an intrathoracic tumor at the base of the left lung, obliterating the costophrenic angle and suggestive of involvement of the diaphragm. There was a foreign body in the right side of the thorax posteriorly. A diagnosis was made of recurring sarcoma in the lower left portion of the chest, probably originating at the site of the previous injury. Operation was advised.

Operation was performed under intratracheal anesthesia on September 27. The scar of the previous operation and a large necrotic area were excised. It was found that the tumor originated in the lower portion of the thorax and

Fig. 25.—Six months after radical removal of the tumor; resection from the seventh to the twelfth ribs with the involved wall of the chest and diaphragm. The patient's general condition was good; there was no return of symptoms and the patient gained 34 pounds.

involved the entire wall of the chest and a portion of the diaphragm. The seventh and eighth ribs had been resected at a previous operation and at that time the reformed bone with a larger portion of the ribs was removed. It was necessary to remove from 10 to 15 cm. from the ninth to the twelfth ribs and from 8 to 10 cm. of the entire wall of the chest with from 3 to 4 cm. of affected diaphragm at its attachment to the wall of the chest. The phrenic nerve on the pericardial pleura just above the diaphragm was then cut (fig. 23). The relaxed diaphragmatic muscle was resutured to the wall of the chest and to the margins of the opening, closing the thoracic cavity from the abdominal cavity. The thorax was closed without drainage. There was considerable shock following the operation and a transfusion was given on the operating table. The pathologist reported endothelioma. The tumor weighed 1,380 Gm.

Convalescence was uncomplicated except for left pleural effusion necessitating repeated aspiration. The patient was given a course of roentgen treatment three weeks following operation. He was dismissed six weeks after operation. The wound was practically healed and his condition was fair. He returned for two courses of roentgen treatment.

Six months after operation the patient's general condition was excellent. Symptoms had not recurred. Roentgen examination of the lungs was negative; there was slight thickening of the pleura. He has gained 34 pounds (15.4 Kg.) and is doing light work (figs. 24 and 25).

CONCLUSIONS

Diaphragmatic paralysis by phrenicotomy is of value as a palliative procedure in certain cases of diaphragmatic hernia in which radical surgical procedures are contraindicated. Preliminary phrenic emphraxis is an aid in the selection of these cases.

Phrenicotomy as preliminary to radical surgical procedure is of value in closing large hernial openings in selected cases. In the most extreme cases, preliminary posterior thoracoplasty may be necessary.

Phrenic emphraxis is of great aid in the technical closure of openings in the diaphragm in operations by the abdominal approach on very obese patients.

Large defects in the wall of the chest and diaphragm may be repaired and a closed thorax maintained by utilization of the paralyzed diaphragmatic muscle following phrenicotomy.

ABSTRACT OF DISCUSSION

DR. LEMON: In the autumn of 1926, I presented to this Society the results of experimental work on dogs, on which unilateral and bilateral phrenic neurectomy had been done. It is known that other planogrades behave precisely as dogs do and it is almost certain that human beings react in the same way in all essential details. They are not seriously affected by unilateral paralysis of the diaphragm and the loss of function of the whole diaphragm is not incompatible with comfortable living.

The physiologic effect of phrenic neurectomy has been checked many times since it was first reported and the results compared with the behavior of man under a similar handicap from loss of muscular function. Patients have been observed who have had true eventration, congenital absence of the hemidiaphragm or diaphragmatic hernia. The essential details of the study of such cases may be condensed by reporting that the operation on animals does not produce either immediate or late functional injury to circulation, digestion or respiration. The animals live normally and exercise in the same way as their normal mates; they are not dyspneic or cyanotic, and their pulse and respiration rates are not increased. The slight reduction in vital capacity is regained promptly and is normal before the surgical wounds have healed. This may be demonstrated by subjecting animals to vigorous exercise in company with normal mates or by measuring the intake of air in liters for a unit of time and comparing with animals of equal weight.

The intrapleural tension is undisturbed from its normal measurements and the readings of the manometer are bilaterally equal when made simultaneously. The loss in function of half or of the whole diaphragm does not affect the function of any other group of respiratory muscles and the excursive movements

of the thoracic arch and of the costal margins remain unaltered. The chest remains bilaterally symmetrical and the excursive movements bilaterally equal in extent. This has been checked time after time by simultaneous kimographic records of the two sides, by actual measurement of the excursion outward on inspiration with the animal conscious, and also with the animal unconscious from anesthesia, and by inspection combined with measurement both before and after stripping the sliding integument and superficial structures from the thoracic wall and actually exposing the ribs and their intrinsic musculature. This procedure has been employed many times immediately prior to killing the animal by overetherization. At necropsy the lungs are not affected by pathologic change within the parenchyma and bronchi or in the blood vessels, and the heart and pericardium are normal. This fact has been demonstrated by microscopic study of the tissue and has been confirmed by C. B. Brown of the Mayo Clinic. The circulation within the paralyzed portion of the diaphragm is unimpaired. The vessels themselves remain normal. The muscle is pale, thin, translucent and the fibers atrophied.

The positive effects are twofold. The long diameter of the thorax is decreased and the musculature of the diaphragm is rendered atrophic. The circulation through the atrophied portion is, however, unchanged and the histologic structure of the vessels is unaltered.

Two men and one woman have been carefully studied after section of the phrenic nerve. They were suitable for study because their chests were symmetric before operation. In all essential details the results were precisely the same as those enumerated. Two details presented slight variation: intrapleural tensions were not determined because tension is not comparable in types that have anatomically different mediastinal partitions, and the paralyzed hemidiaphragm of man rose higher in the thorax than that of the carnivorous animals whose abdominal viscera are relatively less bulky.

This led me to suggest, in 1926, that the operation in man could be used with benefit in general surgery, as well as in its recognized field of thoracic surgery and especially in these instances in which immobility, instead of mobility, is desirable. Thus it seemed to me that it might render operations about the cardiac end of the stomach less difficult technically, that low lesions of the esophagus or of the wall of the chest might be attacked more readily and that tears or rents in the diaphragm might be more easily repaired. Harrington's papers in 1927, and the present report not only confirm the physiologic truths but also have proved the practical surgical importance of the operation beyond the expectation of his associates concerned in purely experimental and clinical work. I think the operation has never before been employed alone for this specific purpose.

In the cases that have been chosen for this report there are two possible methods of procedure, should symptoms persist and require surgical interference: (1) reexploration and sectioning of any portion of the phrenic nerve, which by reason of unusual and atypical origin might have escaped section, and (2) the performance of thoracoplasty so that the thorax may be made immobile and suction of viscera into it prevented. I have experimental evidence that warrants such a procedure. The indication for the first method of attack is easily determined by fluoroscopic observation of the patients.

It seems to me, therefore, that more can be expected in the future. Already success has attended the surgical section of both phrenic nerves in those distressing cases of diaphragmatic tic produced by encephalitis and I am sure that the operation may become more helpful in surgical procedures on the lungs, the abdominal viscera and the central nervous system.

THE DEFENSIVE AND METABOLIC APPARATUS OF THE LUNGS

B. M. Fried, M.D., Boston

In addition to the original articles published in these transactions, there was also presented a paper by B. M. Fried, Boston, The Defensive and Metabolic Apparatus of the Lungs, which has been published in *Archives of Pathology* **6**:1008, 1928.

PROCEEDINGS

SUNDAY, APRIL 29

The Council met at 8:15 p. m., at the Cosmos Club, Washington, D. C.

MONDAY, APRIL 30

The meeting was called to order at 9:30 a. m. by the president, Dr. Evarts A. Graham, at the United States National Museum, Washington, D. C.

The following papers were presented:

Dr. Walter Estell Lee, Dr. Gabriel Tucker and Dr. Eugene Pendergrass, Philadelphia: "Experimental Atelectasis."

Dr. Pol N. Coryllos and Dr. George Birnbaum, New York (by invitation): "Lobar Pneumonia Considered as Pneumococcic Massive Atelectasis: Bronchoscopic Treatment."

Dr. Edward Churchill, Boston: "Pulmonary Vein Occlusion."

Dr. Frank S. Dolley, Los Angeles, and Dr. E. Robert Wiese, Ingomar, Pa. (by invitation): "Effects of a Large, Closed Bilateral Pneumothorax and Thoracic Lymph Flow."

Dr. Howard Lilienthal, and Dr. J. Burns Amberson, New York (by invitation): "Unilateral Pneumothorax: The Behavior of the Mediastinum."

Dr. Stuart W. Harrington, Rochester, Minn: "Phrenicotomy in the Treatment of Diaphragmatic Hernia and Tumors of the Wall of the Chest."

After the noon recess the meeting was continued in the same place at 2 p. m. The afternoon session was devoted to a symposium on "Thoracic Tumors" in which the following essayists participated:

Dr. George J. Heuer, Cincinnati: "Thoracic Tumors."

Dr. J. J. Singer, St. Louis: "Thoracic Tumors: A Roentgen Study."

Dr. Howard Lilienthal, New York: "Cyst of Lung: Recovery Following Operation for Permanent Drainage."

Dr. Leon T. LeWald, New York: "Roentgenologic Diagnosis of Thoracic Dermoids."

Dr. Willy Meyer, New York: "Primary Cancer of the Lung."

Dr. John D. Kernan and Dr. Arthur J. Crancovaner, New York: "Carcinoma of the Lung."

During this symposium the association received the report of the committee appointed in 1927 to consider a National Registry for Thoracic Tumors, and temporarily went into executive session in order to consider this report. The report is set forth in full elsewhere.

TUESDAY, MAY 1

The meeting was resumed in the same place at 9 a. m.

The presidential address was delivered by the president, Dr. Evarts A. Graham, St. Louis, the title being "The Significance of Changed Intrathoracic Pressure."

The following papers were presented as a symposium on the general subject "The Heart and Thoracic Surgery:"

Dr. Paul D. White, Boston (by invitation): "The Relationship of the Heart to Lung Pathology in Disease."

Dr. Alton Ochsner and Dr. George Herrmann, New Orleans (by invitation): "Experimental Surgical Relief of Experimentally Produced Pericardial Adhesions."

Dr. Carl A. Hedblom, Chicago: "Acquired Dextracardia."

Dr. Arthur H. Shipley and Dr. Cyrus F. Horine, Baltimore (by invitation): "Experimental Pericarditis."

Dr. Elliott C. Cutler and Dr. C. S. Beck, Cleveland (by invitation): "Final Results on all Operated Cases of Valvular Disease of the Heart. The Present Status of the Surgical Procedures in Chronic Valvular Disease of the Heart: Final Report of all Surgical Cases."

Dr. A. L. Lockwood, Toronto: "Operations on the Heart."

Dr. T. C. Davison, Atlanta: "Removal of Bullet from the Pericardium Under Local Anesthesia."

After the noon recess the meeting was resumed at 2:30 p. m. at the same place. Discussion on the morning symposium was concluded.

The following papers were presented as a symposium on "Pulmonary Suppuration:"

Dr. Harold Brunn, San Francisco: "Surgical Principles Underlying the One-Stage Operation of Lobectomy."

Dr. H. L. Beye, Iowa City: "Recurring Hemorrhage in Chronic Suppurative Conditions of the Lung: Treatment by Ligation of the Pulmonary Artery; Report of Two Cases."

Dr. Conrad Georg, Jr., Ann Arbor: "Abscess of the Lung Following Fracture of the Ribs."

Dr. Adrian V. S. Lambert and Dr. Carl Weeks, New York (by invitation): "Experimental Production of Abscess of the Lung."

These papers were discussed as a group.

Wednesday, May 2

Dr. B. S. Kline, Cleveland (by invitation) and Dr. Samuel S. Berger, Cleveland (by invitation) (presented by Dr. Young): "Abscesses and Pulmonary Gangrene: Clinical Course and Pathology."

Dr. William Lerche, St. Paul: "Hodgkin's Disease of the Neck and Mediastinum: Bilateral Cervical Operations: Mediastinotomy."

Dr. Frank B. Berry, New York: "Massive Atelectasis Complicating Thoracoplasty for Pulmonary Tuberculosis."

Dr. B. M. Fried, Boston (by invitation): "The Defensive and Metabolic Apparatus of the Lungs: the Lungs and the Macrophage System."

NATIONAL REGISTRY FOR THORACIC TUMORS

At this annual meeting the committee appointed in 1927 to consider the establishment of a National Registry for Thoracic Tumors presented the following report:

Last year a committee was appointed to consider ways and means of establishing a registry for chest tumors. The report of that committee, approved by the Council, will be presented to you this afternoon.

The committee recommends that the registry of chest tumors be an activity of the American Association for Thoracic Surgery, to stimulate the study of chest tumors and to collect data and material in such quantity as to permit a reasonably wide investigation of the clinical picture, pathology, treatment and prognosis of these cases. Relatively rare as some of these tumors are, it has not been possible in the past to gather together data on a sufficient number of cases to allow definite conclusions to be drawn. It is hoped, however, that by pooling the clinical experience of a number of interested clinicians and surgeons this may be rectified in some degree. The data and material of the registry are to be at the service of any one participating in the investigation of such cases, or of accredited and interested individuals.

The committee further recommends as follows:

That the records be housed in fireproof files in the Cincinnati General Hospital.

That the Council be empowered to appoint a committee, to consist of five surgeons, three pathologists, two internists, one roentgenologist, one bronchoscopist and a registrar.

That the registry shall include all primary intrathoracic tumors except aneurysms, and all primary tumors of the thoracic wall that impinge on the pleura.

That the registry committee be empowered to adopt a classification and formulate record forms to insure uniform reporting of the material.

That only such cases shall be included as are accompanied by ample and accurate data, the inclusion of a given case to be decided by the committee.

That solicitation for registration be confined at first to the members of the American Association for Thoracic Surgery, the American Surgical Association, the Association of American Physicians, and Pathologists of the prominent clinics of the country.

As to the financial support of the registry, the committee recommends that the Association shall assume the annual expenses incurred in connection with the registry, not to exceed $250, as a legitimate charge against the income from endowment, and if such income be insufficient, the members of the Association support the registry by voluntary contributions.

It was regularly moved and seconded that this report be adopted. The motion was unanimously carried.

In accordance with the foregoing motion the following committee was appointed:

George J. Heuer, M.D., Chairman, Cincinnati
William DeW. Andrus, M.D., Registrar, Cincinnati
Edward W. Archibald, M.D., Montreal
N. C. Foot, M.D., Cincinnati
Evarts A. Graham, M.D., St. Louis
Carl A. Hedblom, M.D., Chicago
Willis S. Lemon, M.D., Rochester, Minn.
Howard Lilienthal, M.D., New York
Leon T. LeWald, M.D., New York
Frederick T. Lord, M.D., Boston
Grabriel Tucker, M.D., Philadelphia

CHANGES IN CONSTITUTION AND BY-LAWS

At the Eleventh Annual Meeting of the American Association for Thoracic Surgery, Article VII. of the By-Laws was amended to read as follows:

Article VII

Sec. 1.—Honorary members of the Association are exempt from all initiation fees, dues and assessments.

Sec. 2.—Annual dues for Active Members shall be $20.

Sec. 3.—Annual dues for Associate Members shall be $10.

Sec. 4.—Annual dues for Senior Members shall be $10.

Sec. 5.—Initiation fee for those elected directly to active membership shall be $25.

Sec. 6.—Initiation fee for those elected to Associate Membership shall be $10. If and when an Associate Member is elected to Active Membership he shall pay an additional $15 initiation fee to Active Membership.

Sec. 7.—In illustrating the annual transactions the Association will incur expenses not to exceed $15 per article. If the cost for illustrating any article exceeds the sum of $15 the excess shall be charged as a special assessment against the member concerned.

Sec. 8.—Income from the Endowment Fund shall be expended as the Council directs.

These changes become immediately effective in regard to newly elected members. They will become effective Jan. 1, 1929 in regard to all other members.

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